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CANCER OF THE EAR.

A Report of 150 Cases.*†‡

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Cancer is the end result of mutational processes, which giving rise to new races of cells, do not have the capacity to mature or to differentiate completely in response to regulatory host mechanisms.[1] Cancer of the ear has the same biological characteristics as cancer found at other sites in the human body. Cancer involvement of an organ of special sense, with the proximity of dura and brain, cranial nerves, temporomandibular joint and zygoma presents a formidable operation, for not only may hearing be lost and balance disturbed, but the incidence of postoperative intracranial infection complicates a hazardous surgical resection; therefore, it seems important at this time to review the diagnosis and surgical managament of the various types of cancer of the ear, as seen in 150 patients over a period of 15 years.

The objectives of this report are to analyze these cases of aural cancer, with particular reference to etiology and pathology, to classify them according to therapy indicated, and to describe the surgical procedure employed for each classification.

An early, excellent review of the literature was presented

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by Peele and Hauser in 1941.[2] According to these authors, Wilde and Roudot, Schwartze, Lucae, Kidd, and others, in the period around 1775, were the first to discuss carcinoma of the middle ear. In 1883, Politzer[3] gave a comprehensive description of cancer of the ear, as manifested in his patients. Kretschmann,[4] in 1886, gave a comprehensive description of reported cases, and included four of his own. Between 1804 and 1899, 121 otitic tumors were recorded, according to Zeroni[5]; and Newhart[6] found 34 cases of carcinoma of the middle ear reported between 1899 and 1917 (only eight of the 34 cases being reported in American medical journals). In 1921, Broders[7] made a statistical analysis of 63 cases of epithelioma of the ear, which was the first comprehensive article published in the English language. Yates[8] in 1936, published his findings covering a thorough search of the literature; he listed 14 cases reported between 1917 and 1924, and 24 cases between 1924 and 1936. In 1942, Figi and Hempstead[9] reported 48 malignant tumors of the middle ear and mastoid from 1922 to 1941, as well as 500 others involving the pinna or external auditory canal. Morrison,[10] in 1946, reviewed 13 cases of malignant tumors of the ear, including the external ear. Grossman, Donelly and Snitman,[11] in 1947, reported six cases of squamous cell carcinoma of the middle ear and mastoid. Garnett Passe[12] reported a case of primary carcinoma of the Eustachian tube in 1948, and reviewed two other cases in the literature. In 1950, Towson and Shofstall,[13] in an excellent review, analyzed seven cases of ear cancer, from the Jefferson Hospital, Philadelphia. Mattick and Mattick,[14] in 1951, discussed their experience with ten cases of cancer of the middle ear and mastoid. In 1954, Figi and Weisman[15] describing a wide experience with the surgical management of ear cancer and chemodectoma at the Mayo Clinic, cited 124 cases of cancer of the middle ear and mastoid covering the period from January, 1907, through December, 1951.

In 1954 Parsons and Lewis,[16,17] encouraged by the partial success of Ward, et al.,[18] and Campbell, et al.,[19] in resecting the temporal bone piecemeal for carcinoma, demonstrated the feasibility of a combined intracranial, extracranial approach in a one stage *en bloc* subtotal resection of the temporal bone

for carcinoma involving the middle ear and mastoid. In 1958 these same authors reported successful temporal bone resections in 27 cases with an expected fair salvage rate.

Meanwhile, in 1955 Miller[20] analyzed 88 cases of carcinoma of the external auditory meatus. He emphasized the unpredictability of these lesions as to local extent and recurrence regardless of histologic type, and demonstrated the lymphatic drainage of the area.

DEFINITION.

Cancer of the ear is a term covering a multiplicity of lesions, ranging from cancer of the skin of the auricle to malignant tumor which erodes the petrous portion of the temporal bone. The problems of the various lesions differ considerably, except in cases of advanced lesions (where the temporal bone is invaded) for these, radical measures are mandatory, whatever the site of origin may be. Cancer of the pinna and cancer of the auditory canal are designated as "early" (below 2.5 cm.) and "advanced" respectively, because of the difference in therapy required. Cancers of the middle ear, of the mastoid, and of the petrosa, at any stage, require radical surgery and are grouped in a common therapeutic approach.

INCIDENCE.

According to published reports, cancer of the ear is a rare condition; and even a busy otolaryngologist may see, at most, only one or two ear cancers in his entire career. Figi and Weisman,[15] in analyzing the records of the Mayo Clinic for 45 years (January, 1907, through December, 1951), found only 124 cases of cancer of the middle ear and mastoid. By way of contrast, more than 13,000 patients were treated for cancer of the stomach at that clinic in the same period. Among 212,000 cases of aural disease seen between 1905 and 1924, at the Manhattan Eye, Ear and Throat Hospital, Robinson[21] found a diagnosis of tumor in only 48 cases, or a ratio of 1:4000. Schall[22] found that in a 12-year period, at the Massachusetts Eye and Ear Infirmary, only 15 patients out of 90,040 with pathological conditions of the ear, had neo-

plasms. Furstenberg[23] found cancer of the middle ear in only two out of 40,000 patients in the Department of Otolaryngology at the University of Michigan Medical School. Tod,[24] quoting from the records of the London Hospital, where 200,000 cases are seen annually, found only one case. Lodge and his co-workers[25] found six cases of cancer of the temporal bone reported in a population of one million people, and estimated that 0.006 per thousand of living persons suffer from aural cancer at a given time. Mattick and Mattick[14] reported a diagnosis of intrinsic cancer of the ear in ten out of 35,000 tumor cases at the Roswell Park Memorial Institute in Buffalo. Towson and Shofstall[13] point out that the proportion of cancer of the middle ear and mastoid to all otologic pathological conditions is 1:5000 to 1:20,000.

In their report of a 25-year study, Driver and Cole[26] note that the ear was involved in 5.5 per cent of all the patients with skin cancer. Labarde and Via[27] state that 8 per cent of all skin cancer originates from the auricle. Broders[7] had estimated some years earlier that 84 per cent of aural cancer involved the auricle, 12 per cent the external auditory meatus, and 1.5 per cent the middle ear and mastoid. In the group of 150 patients discussed here, the origin of the cancer was the auricle in 60 per cent, the external auditory canal in 28 per cent, and the middle ear and mastoid in 12 per cent.

Conway and Howell[28] note the high incidence of auricular cancer in blond persons, and do not report a single case of it in a series of 100 Negro patients. Not a single case of cancer of the auricle in a Negro occurred among the author's patients, although two Negro patients had cancer of the middle ear.

ETIOLOGY.

Sex, age, heredity, and chronic infection are the main factors that influence the development of cancer of the ear. Cancer of the auricle primarily affects the aged, most often the male, and is most likely stimulated by environmental factors. Actinic rays of the sun, frostbite, insect bites and

trauma have all been responsible for cancer of the external ear in our patients.

Lodge[25] and his associates consider that cancers of the middle ear and of the ear canal are comparable to Marjolin's ulcer, with its chronic sanious discharge producing cellular irritation that develops into cancer. Chronic otitis externa and chronic otitis media have also been forerunners of cancer in some of our patients, although in most cases there was no history of pre-existing infection. Coachman[29] has reported a patient with squamous cell carcinoma of the middle ear secondary to cholesteatoma; and Sparks[30] had one with carcinoma, who had long-standing otitis externa. Barnes[31] believes that a large number of cancers probably originate in the lining of mastoid cavities, such lining having become altered by prolonged suppuration. Hyperkeratosis, too, has frequently appeared to be a beginning of cancer of the ear.

Towson and Shofstall[13] and Fredricks[32] consider trauma and chronic irritation as etiological factors. They feel that chronic eczema, dermatoses, frostbite, keratosis, sebaceous cysts, papilloma, cutaneous horns, psoriasis, lupus vulgaris, xeroderma pigmentosum, burns, razor cuts may all contribute to the development of cancer. Trauma caused by the pressure of eyeglasses is included among these factors.

Ackerman and del Regato[33] reported that out of 32 of their patients with ear cancer, only three were women; however, in our patients, as will be demonstrated, the sex incidence varied with the anatomical site of the primary lesion.

Driver and Cole[26] found 64.3 years to be the median age of patients with cancer of the ear, and Broders[7] found it to be 61 years. In our patients, the median age for all was 63 years; 72 years was the median age for those with cancer of the auricle, and 55 years for those with cancer of the auditory canal, of the middle ear, and of the mastoid.

PATHOLOGY.

In 1956, Schenck[34] presented an excellent classification of the various pathological types of malignant ear tumors. Ac-

cording to this classification, the present author has grouped the various types of tumors found in his patients, as listed below:

I. Epithelial Tumors:

A. Malignant Surface Epithelial Tumors:

1. Epithelioma:

a. Squamous Cell Carcinoma:

Broders,[7] Towson and Shofstall,[13] Driver and Cole,[26] Conway and Howell,[28] Brunner,[35] Eller and Eller,[36] all consider squamous carcinoma the most common type of cancer found in the ear. Although in our own patients only one-third of the lesions arising on the auricle were squamous cell, 75 per cent of the advanced cancers of the auditory canal and middle ear were of this histologic type. Eleven per cent of these tumors had metastasized to the neck nodes by the time of the patient's admission.

b. Basal Cell Carcinoma:

In conformity with the type of cancer most often found elsewhere on the skin surface of the body, two-thirds of the cancers in this author's patients, arising on the auricle, were basal cell carcinomas. About 50 per cent of the early, and 11 per cent of the advanced auditory canal lesions were of this type. In five of the patients, the lesions fungated from the auricle, and invaded the middle ear, mastoid, and squamous portion of the temporal bone.

B. Malignant Glandular Epithelial Tumors:

1. Adenocarcinoma:

This tumor may arise from sebaceous glands or ceruminous glands of the ear canal. Schall,[22] Furstenberg,[23] Lukens,[37] Grabschied,[38] Fraser,[39] Thorell,[40] and Fabre,[41] all reported cases of this type of cancer. In 1941, Warren and Gates[42] described the first case of carcinoma of the ceruminous glands. In four of the author's patients the lesion developed in the auditory canal, and two were diagnosed as originating in the

ceruminous glands. Two others fungated into the middle ear from a parotid primary.

II. Mesenchymal Tumors:

Sarcoma:

Cases of embryonal rhabdomyosarcoma of the temporal bone have been reported by Blanchard and House,[43] Karatay,[44] and Holman.[45] According to Stobbe and Dargeon[46] the median age of patients with this tumor is 6.1 years. The tumor is always locally invasive, metastasizes distantly by blood and lymph routes but does not involve the regional lymph nodes. Only one of the patients in the present author's series, a five-year-old boy, was found to have this condition.

Soft parts sarcomas are not usually expected in this anatomical site. One of the rare cases in this series was a spindle cell sarcoma arising in the middle ear of a 43-year-old white female.

III. Malignant Tumors of Questionable Origin:

Malignant Melanoma:

A comprehensive report of 36 patients with melanoma of the external ear, treated between 1928 and 1934, is presented by Sylvan and Hamberger[47] from the Radium Hemmett in Stockholm. The median age on admission from this group of patients (consisting of two children, ten and 12 years old respectively, and 34 adults) was 55 years. Of the 36 patients, 21 were male and 15 female. Two additional cases of melanoma of the ear are reported in the literature: one by Friedmann and Radcliffe,[48] and the other by Cordes and Masing.[49]

Our series includes two cases of melanoma arising from the posterior surface of the auricle, and measuring less than 2 cm. One of these patients had proven positive neck nodes on admission. Another arose in the auditory canal and had neck node metastases. A nine-month-old infant had a melanoma of the middle ear. This was undoubtedly due to a transplacental implant from the mother, who subsequently died of widespread melanomatosis.

CLASSIFICATION BY THERAPEUTIC MANAGEMENT.

Early Cancer of the Auricle: (70 Cases).

These skin lesions, by definition, are cancers that are confined to the pinna, that have not spread to adjacent structures and measure under 2.5 cm. Seventy of our adult patients were afflicted by 73 tumors of this type. The right ear was involved more frequently than the left, and in three cases, both ears were affected coincidentally (see Table I).

TABLE I.

Early Cancer of the Auricle (Under 2.5 cm.).

Pathological Type	No.	Median Age	Male	Female	Surgery	Radiation	Untreated
Basal Cell	46	73	0	4	46	0	0
Squamous Cell	23	71	0	1	23	0	0
Melanoma	1	55	1	0	1	0	0
Total	70	70	1	5	70	0	0

Age and Sex. Cancer of the skin seems to affect older people predominantly. In our group of 70 patients with this disease, the median age was 70 years. The youngest patient was 45 years old, and the oldest was 89.

This condition rarely affects females. Of all our patients with early cancer of this type, 92 per cent were males.

Location. We found the most common site of origin to be the helix in our patients, 50 per cent of all the lesions occurring at that location. The central region of the pinna (concha, triangular and scaphoid fossae, antihelix and its crus) was the second most frequent site, and accounted for 30 per cent of all the lesions. Twelve per cent of all the lesions arose from the posterior surface of the auricle, and the remainder was located on the concha, lobule, and pre-auricular region, in that order (see Fig. 1).

Pathology. In 67 per cent of our cases, the early lesions on the external ear were basal cell carcinomas. (These lesions are locally invasive and slow-growing, and do not, as a rule, metastasize.) In 30 per cent, the lesions were squamous cell

carcinoma. We also have one case of malignant melanoma arising on the posterior surface of the auricle.

Symptoms. The majority of our patients were asymptomatic; however, in most cases, the lesions when present, were obvious to the patient or were found by the examining physician. A few of the lesions became ulcerated and occasionally got infected with subsequent bleeding and pain.

Treatment. Local medical treatment in the form of salves and caustics had been applied, before admission, to most of

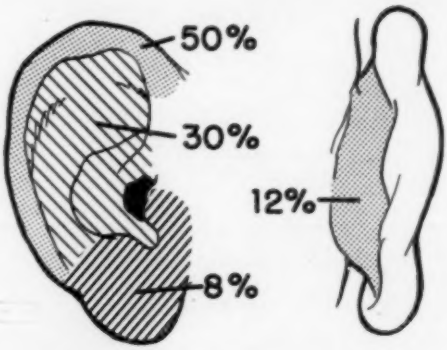
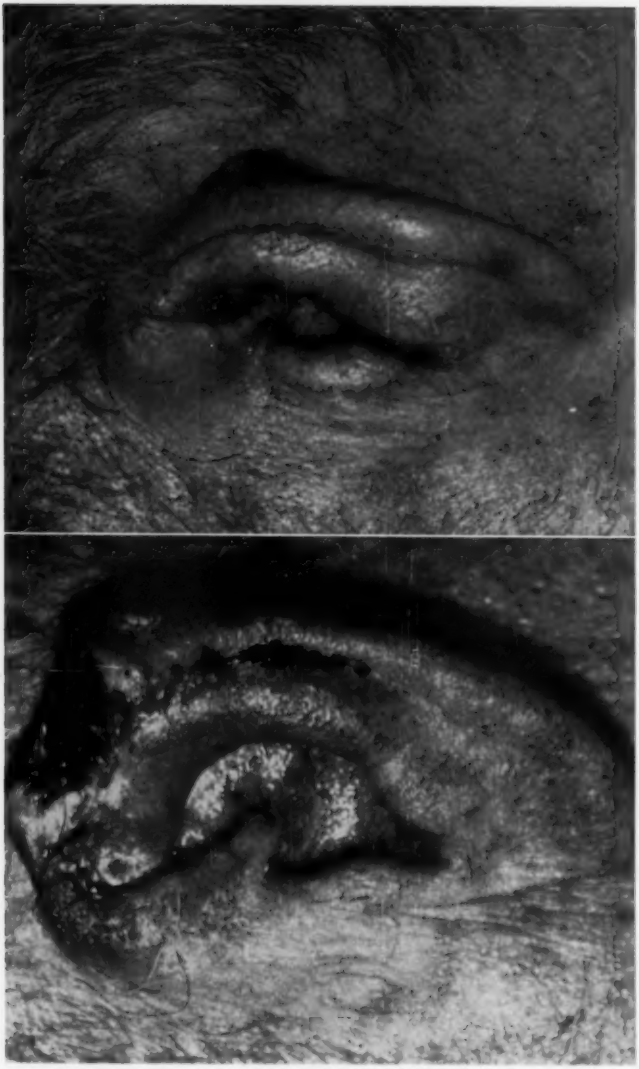


Fig. 1. Distribution of lesions arising from the auricle.

the lesions for periods ranging up to five years. Five patients had been given X-ray treatment, two had been subjected to electrocauterization, two had been surgically treated and had had a recurrence, and one case had had both surgery and radium treatment.

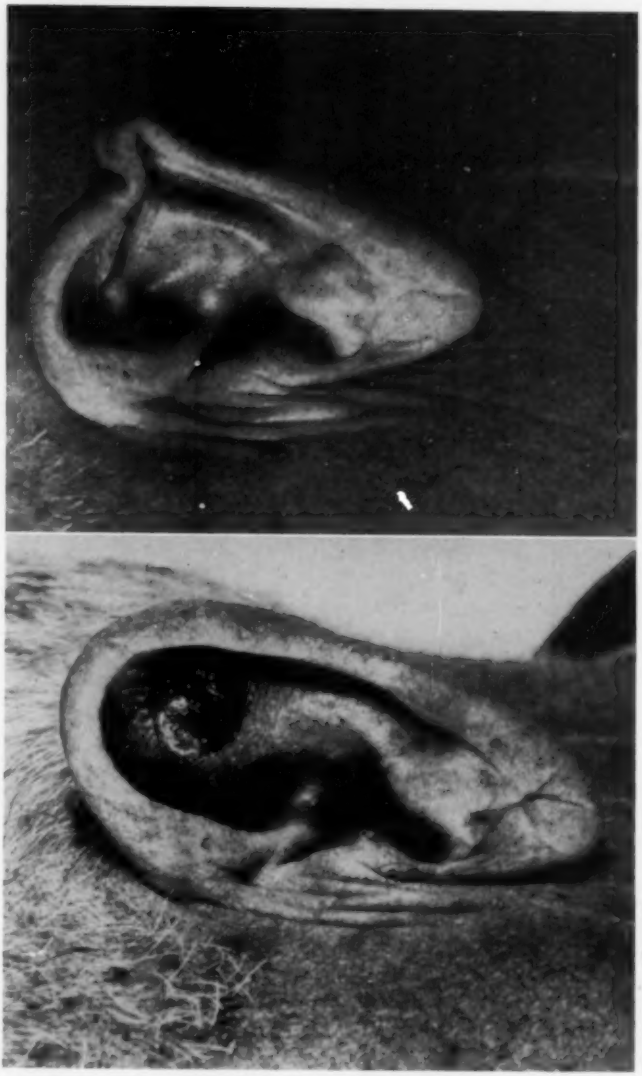
Some authors advocate radiation for patients with ear cancer[26,33]; however, radiation chondritis and necrosis of underlying bone may be serious complications. Chemosurgery, preferred by Mohs,[50] is not the treatment of choice. The procedure that has proven uniformly successful is wide excision, without regard to cosmetic effect. The good results, as reported by Fredricks[32] and Conway and Howell,[28] give



b. Postoperative.

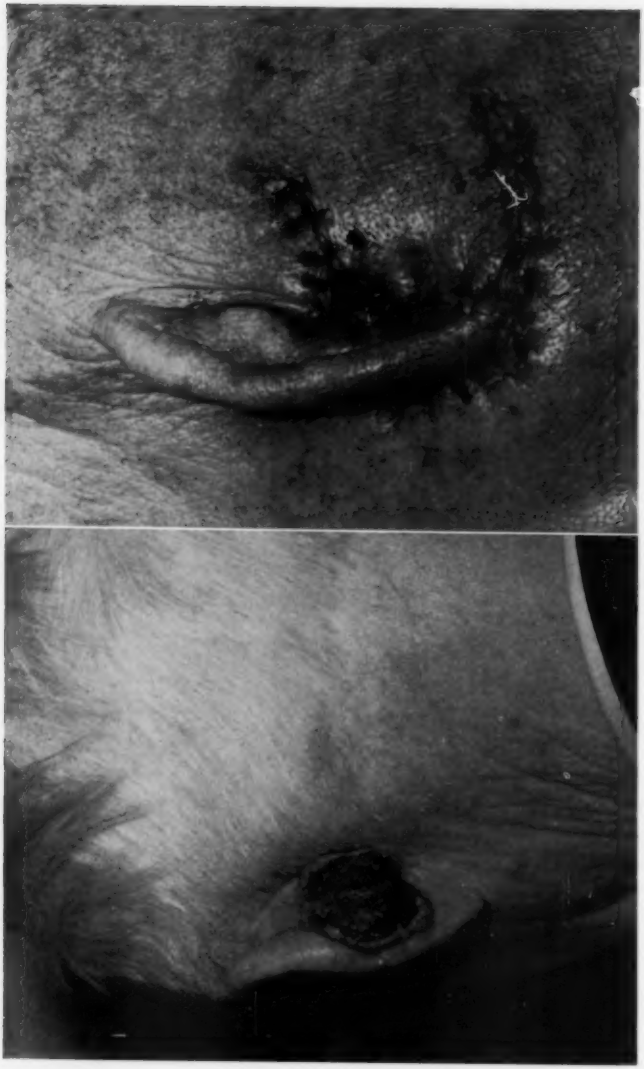
a. Preoperative.

Fig. 2. Excision of cancer of the helix.



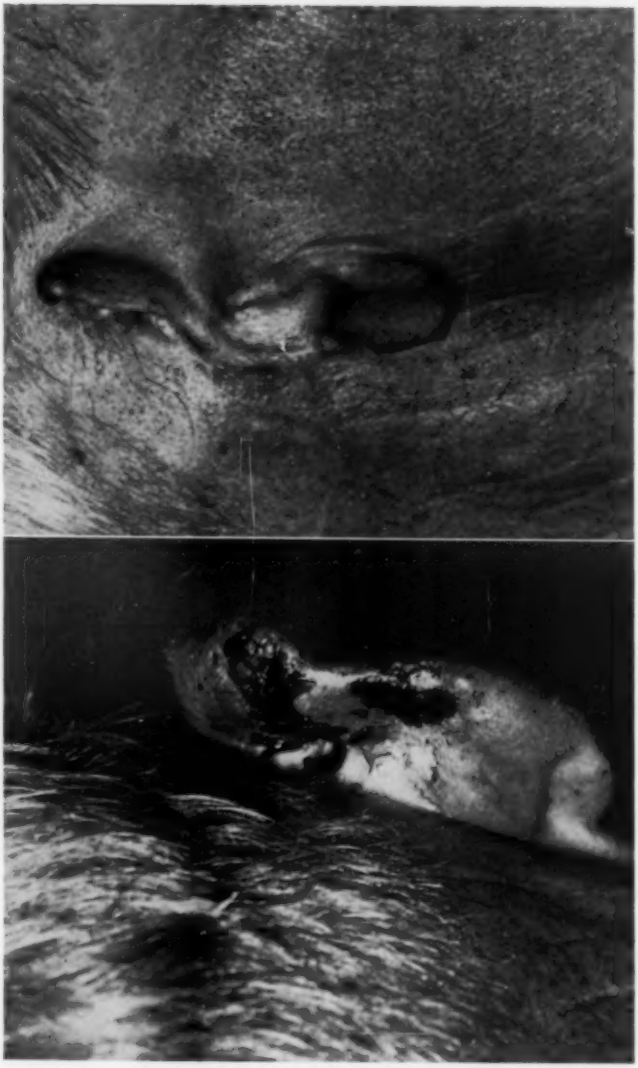
a. Preoperative. b. Postoperative.

Fig. 3. Full thickness excision of cancer of the central portion of the auricle.



a. Preoperative. b. Postoperative.

Fig. 4. Excision of lesion of posterior surface of ear with closure by scalp flap.



a. Preoperative. b. Postoperative.

Fig. 5. Excision of major portion of auricle with extensive cancer.

support to the excellent prognosis in patients treated by this method.

Sixty-seven per cent of our patients were treated by wedge excision and primary closure. Both skin and cartilage were removed, giving very satisfactory cosmetic results (see Fig. 2). Twenty per cent were treated by wide local excision and full-thickness skin graft; in these cases, the lesions did not

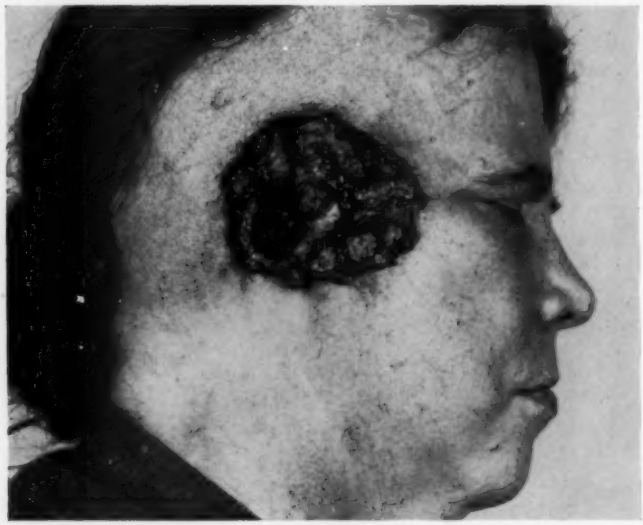


Fig. 6. Advanced cancer involving the entire pinna and adjacent areas.

lend themselves readily to primary closure, as those of the concha (see Fig. 3). In three cases, the posterior lesions were excised and a temporal rotated scalp flap was made to cover the defect (see Fig. 4). In three cases, a wedge excision with homolateral neck dissection was performed, as a primary method of treatment. The larger lesions may at times require sacrifice of a major portion of the auricle, as evidenced in one case (see Fig. 5).

Results. As indicated previously, the median age for this

group of 61 patients was 70 years. Because of the advanced age of these patients, 80 per cent were either lost to follow-up or died of other diseases. For these reasons, this group cannot be analyzed statistically for a five-year salvage rate. Those patients who were followed appeared to be uniformly free of recurrent cancer, during the period of follow-up.

Advanced Cancer of the Auricle: (20 Cases).

Once the cancer extends beyond the limits of the auricle and involves the middle ear, temporal bone, parotid gland, and mandible, serious problems arise. Of our entire group of 150 patients, 20 suffered from this type of advanced disease and

TABLE II.

Advanced Cancer of the Auricle.

Pathological Type	No.	Median Age	Male	Female	Surgery	Radiation	Untreated
Basal Cell	12	70	8	4	7	3	2
Squamous Cell	7	73	6	1	5	0	2
Melanoma	1	55	1	0	1	0	0
Total	20	70	15	5	13	3	4

were treated by various methods, as discussed below (see Fig. 6 and Table II).

Age and Sex. The median age for this group was 72 years, with about the same distribution as in the group with early lesions.

Fifteen of the 20 patients were male, and five were female.

Pathology. Seven out of this group of patients had squamous cell carcinoma; of these, two had neck nodes requiring a homolateral neck dissection. Twelve others had basal cell carcinoma, one of which metastasized to neck nodes. One case was a melanoma with cervical metastasis.

Symptoms. All the patients gave a history of a sore developing on the auricle and progressing for a period of one year or more; the median period was 40 months. Eight patients had had previous limited surgery or X-ray treatment,

or both. Pain did not appear to be a factor until bone erosion occurred, leading to secondary infection.

Treatment. Of the 20 patients with advanced cancer, three refused treatment, one was 90 years old (who died before any treatment was administered), three were given palliative X-ray therapy because of feebleness and advanced age, and the remaining 13 were treated by radical surgery.

Excision of the auricle, mastoidectomy, resection of the ear canal, and parotidectomy with skin graft were done in eight of the 13 surgical cases. Four of these eight patients also had dissection of the homolateral neck, as part of the operation, and a third had radon seeds implanted into the residual disease, on the dural plate. In the five other surgical cases, excision of the auricle and subtotal resection of the temporal bone were carried out, by the technique of Parson and Lewis.[16] In two of these cases, the defect was replaced by a rotated scalp flap.

Results. The three patients who were given X-ray therapy died within six months to two years following treatment.

Of the eight patients treated by excision of the auricle, mastoidectomy, resection of the ear canal, and parotidectomy with skin graft, six died of disease within a period of eight months. The seventh patient has been free of disease for five years, and the eighth was lost to follow-up after staying free of disease for four years.

Of the five patients subjected to excision of the auricle and subtotal resection of the temporal bone (see Fig. 7), two have gone free of disease for five years, two (recently operated) have been free of disease for periods up to two years, and one died two months after surgery, due to a brain hemorrhage. One of these seven patients developed paralysis of the vagus nerve, and required tracheotomy and a feeding tube for two months after surgery.

Cancer of the External Auditory Canal: (42 Cases).

In 42 of our patients, the cancer originated in the external ear canal. Seven cases had early lesions confined to the

membranous portion of the auditory canal; and 35 had advanced lesions invading the middle ear, mastoid, and contiguous structures.

Age and Sex. The youngest of these patients was 24 years old, the oldest was 75, and the median age for the entire group was 55 years.

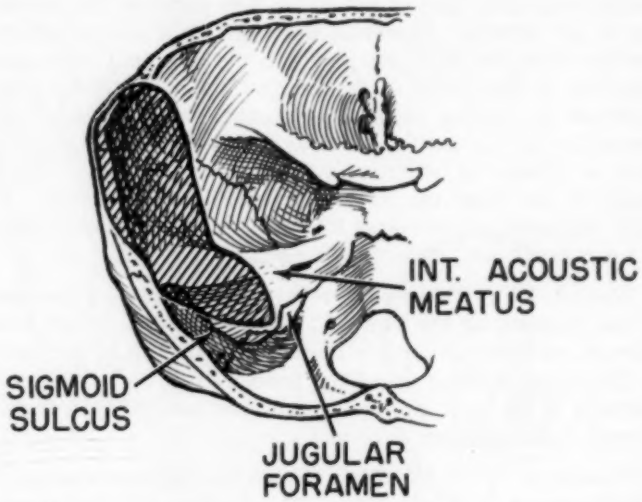


Fig. 7. Extent of bone removed in subtotal resection of temporal bone.

Twenty-four patients were female, and 18 were male. This proportion is the reverse of that found in patients with cancer of the auricle, as previously indicated.

Pathology. In 69 per cent of our patients, the cancer originated in the ear canal and was diagnosed as squamous cell epithelioma. Of all the early cancers among our patients, only 27 per cent were of this type; and of the advanced cancers, 77 per cent were squamous cell carcinomas. In three of the latter group, proven positive neck nodes were present at the time of admission. Only 20 per cent of all our patients had basal cell carcinomas. The basal cell carcinomas com-

prised 50 per cent of the early ear canal lesions, and 11 per cent of the advanced lesions. Four of the 42 patients had adenocarcinoma, which, in two appeared to arise from the ceruminous glands. One patient of the 42 had advanced malignant melanoma, and died of disease two years after surgery (see Table II).

Symptoms and Signs. The patients with early cancer reported occasional pain in the canal, that persisted for periods up to six months. Otorrhea, deafness, pain and occasional vertigo were the usual symptoms in the advanced cases, and reported in that order of frequency. These symptoms were reported as lasting from four months to three-and-a-half years, and for six months on the average. Five of the patients gave a history of chronic otitis of many years' duration. Palsy of the facial nerves was present in four patients. In most instances, an ulcerated bleeding lesion was found filling the ear canal, and often involving the middle ear.

Positive X-ray findings were present in only 50 per cent of the patients, at the time of biopsy. Of these, 30 per cent showed evidence of chronic mastoiditis, and in 12 per cent, a tumor and destruction of the bone were present. The remaining 8 per cent showed a previous mastoid defect and chronic inflammation.

Treatment. Nine out of the 42 patients had been treated by irradiation, prior to admission. Three others had had a mastoidectomy followed by Roentgen therapy; two had had mastoidectomy alone; and the remainder had had no treatment at all. Six patients with *early* lesions involving only the membranous canal were treated by a sleeve resection of the ear canal. Another was treated elsewhere. The incision employed in this procedure is indicated in Figs. 8-a and 8-b.

The incision consists of the upper limb of a parotidectomy incision (as described by Martin[51]) which is deepened so as to expose the inferior portion of the external auditory canal. Care is taken to avoid the facial nerve, and it may be necessary to expose it in a deep-seated lesion, so that it may be retracted and avoided in the subsequent surgery. The remaining portion of the auditory canal is excised through an

endaural incision much like coring an apple, and the entire ear canal is removed. The defect is grafted with a split-thickness graft, sutured to the endaural incision and maintained in place by a stent.

Three of the six patients treated by this method have been free of disease for four years and two others have had no recurrence for three years (see Table III).

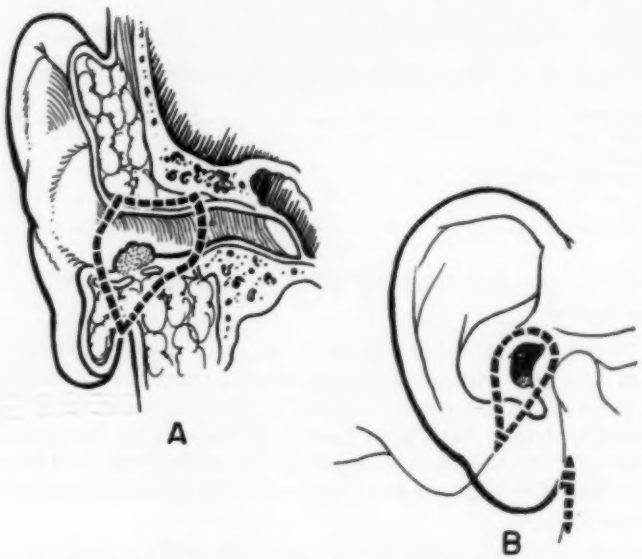


Fig. 8-a. Extent of excision of soft tissue for early auditory canal lesion.
Fig. 8-b. Area of excision, lateral view.

Of the 35 patients with *advanced* lesions, 14 were subjected to a radical mastoidectomy, and to a parotidectomy in addition, as required. The same procedure, together with a homolateral dissection of the neck, was used for two patients of the 14 in whom proven positive neck nodes were present. One of these two patients had a recurrence following neck dissection and mastoidectomy, was given irradiation, and has

been free of disease for five years. The remainder of the patients (13 cases) who had been operated on by this method were failures, were given X-ray treatment, and died of the disease within a period of three months to two years. In this last group, there were two patients with adenocarcinoma, and one with melanoma; the others all had squamous cell carcinoma. Of the entire group of 35 patients, one other had a radical mastoidectomy at another hospital, and has had no recurrence for 18 years. Both of the successfully treated

TABLE III.

Early Cancer of the Auditory Canal.

Pathological Type	No.	Median Age	Male	Female	Surgery
Basal Cell	4	51	0	4	4
Squamous Cell	2	57	1	1	2
Adenocarcinoma	1	48	1	0	1
Total	7	55	2	5	7

patients, one male and the other female, had squamous cell carcinoma.

Four patients, out of a group of 35, were treated by irradiation, and died within six months after therapy was instituted.

Thirteen patients were treated by subtotal resection of the temporal bone; have been free of disease up to the present time, and one has had no recurrence for more than five years. Two out of this group of 13 patients died of complications of surgery.

In three patients out of the group of 35, a temporal craniotomy was performed and because the disease was found to involve the dura, brain, and base of the skull, postoperative irradiation was also administered. All three patients succumbed to disease within a year after treatment (see Table IV).

Cancer of the Middle Ear and Mastoid: (18 Cases).

This group of 18 patients consisted of two with primary cancer in the mastoid, and 16 with primary cancer in the middle ear (see Table V). Glomus tumors are omitted be-

cause of their uniqueness and discussion by the author in another paper.

Age and Sex. The age span was nine months to 76 years, with a median age of 55 years.

Nine patients were female and nine were male. Both mastoid conditions involved the males.

TABLE IV.

Advanced Cancer of the External Auditory Canal.

Pathological Type	No.	Median Age	Male	Female	Surgery	Radiation	Untreated
Basal Cell	4	47	3	1	4	0	0
Squamous Cell	27	55	9	18	23	4	0
Adenocarcinoma	3	54	3	0	3	0	0
Melanoma	1	74	1	0	1	0	0
Total	35	55	16	19	31	4	0

TABLE V.

Cancer of the Middle Ear and Mastoid.

Pathological Type	No.	Median Age	Male	Female	Surgery	Radiation	Chemotherapy
Squamous Cell	12	57	6	6	9	3	0
Melanoma	2	32	1	1	0	1	1
Embryonic Rhabdomyosa.	1	5	1	0	1	0	0
Spindle Cell	1	43	0	1	1	0	0
Adenocarcinoma	2	45	1	1	2	0	0
Total	18	55	9	9	13	4	1

Pathology. The pathology in this group of 18 patients was distributed as follows: 12 had squamous cell carcinoma (these included both patients with primary cancer in the mastoid); two patients (one infant and one elderly male) had malignant melanoma; one had spindle cell sarcoma; and one (a five-year-old child) had embryonal rhabdomyosarcoma. Two patients had adenocarcinoma extending from the parotid gland.

Symptoms. Of this entire group of patients, 40 per cent gave a history of otorrhea of one to 50 years' duration, in some cases dating back to early childhood. The symptoms most often reported were bleeding from the ear (lasting six

weeks to one year), pain (for periods of two to 12 months), and progressive deafness. Vertigo was reported by two patients, and facial palsy by three. Pain and swelling over the mastoid process were reported by both patients with cancer of the mastoid.

On admission, one patient, with malignant melanoma, had proven positive neck nodes; bone destruction was evident on X-ray examination in 40 per cent of this group; 50 per cent showed pathological changes suggesting chronic inflammation; one had only a mastoid defect following surgery; and in one case, the X-ray findings were negative.

Treatment and Results. Nine of these 17 patients had had no previous treatment; nine had had a mastoidectomy, or a mastoidectomy and Roentgen therapy.

On admission, subtotal resection of the temporal bone was performed on 13 patients. Of these 13, four are still alive, free of disease—one, operated on for spindle cell sarcoma, has survived for six years, one for four years, and two for three years. Nine were failures. Three patients developed meningitis after surgery. One patient died of pneumonia associated with Xth nerve paralysis.

One other of the group of 18 patients was found to be unresectable due to involvement of the brain and base of the skull, was given postoperative betatron irradiation and ended in failure.

Four patients of the group of 18 were treated by irradiation only, and died of disease. The infant with melanoma was treated with nitrogen mustard and also succumbed to the cancer.

DISCUSSION.

For practical purposes cancer of the ear has been divided into *A*. Cancer of the auricle, *1*. early, and *2*. advanced; *B*. Cancer of the external canal, *1*. early, and *2*. advanced; *C*. Cancer of the middle ear and mastoid.

Though seldom seen in office practice, the management of

an ear cancer case is a dramatic episode for any otologist. Based on the wide clinical measurement of Towson and Shofstall,[12] cancers of the middle ear and mastoid are indeed rare, and occur in 1:5,000 to 1:20,000 otologic pathological conditions. Cancer of the auricle comprises about 5.5 per cent to 8 per cent of all skin cancers. The author, in assaying a group of 150 cases, has found the origin of the cancer was the auricle in 60 per cent, the external canal in 28 per cent, and the middle ear and mastoid in 12 per cent. Broders[7] reported a high proportion of cancer originating on the auricle (84 per cent) which may represent a truer indication of lesions seen at a general hospital, as against the experience at a cancer institution (where referred treated failures are more commonly seen). No case of auricular carcinoma has been reported in a Negro, although the author has cited two Negro patients with cancer of the middle ear. Sex, age, heredity and chronic infection are the main etiological factors. Cancer of the pinna affected males in 92 per cent of the early cases and 75 per cent of advanced cases. By contrast, cancer of the auditory canal was more common in females, and was distributed equally between the two sexes in the middle ear. The median age of those with cancer of the auricle was 70 years; that of the ear canal and middle ear and mastoid was 55 years. Cancer involving the Eustachian tube was invariably an extension from the nasopharynx and has been excluded from this study. Consistent with experience elsewhere on exposed skin surfaces of the body, two-thirds of the cancers of the auricle were of the basal cell type. Almost one-third were of the squamous cell variety and in two cases neck node metastases were present on admission. By contrast, three-fourths of the widely invasive lesions of the auditory canal and middle ear were of the squamous cell type and 11 per cent of these tumors had metastasized to neck nodes by the time of the patient's admission. Adenocarcinoma originated in the auditory canal, and in two of four cases of this series, appeared to arise in the ceruminous glands. Malignant melanomas were located on the posterior surface of the auricle, the auditory canal and the middle ear with a tendency to cervical metastases. Melanoma at this site appears to be most aggressive regardless of the size of the primary lesion. An

additional embryonal rhabdomyosarcoma of the middle ear in a five-year-old boy is included in this study. A spindle cell sarcoma arising in the middle ear of an adult white female adds to the collection of this rare histologic type.

Pain is an uncommon symptom in ear cancer until infection and bony invasion takes place. The diagnosis of cancer of the auricle is obvious to the examiner and confirmed always by biopsy. Chronic otorrhea, hemorrhage, mastoid swelling, facial paralysis, tinnitus, deafness and vertigo are symptoms and signs of advanced ear canal middle ear and mastoid lesions. Roentgen evidence of temporal bone destruction comes late and augurs poorly for resectability of an ear cancer.

The importance of classification by therapeutic approach must be emphasized. Each anatomical site and extent of the primary lesion is a test of the versatility and experience of the cancer surgeon. Radiation and chemosurgery are fraught with complications of necrosis and infection and were not used primarily as therapeutic modalities by the author.

Early lesions of the pinna (2.5 cm. or less) are usually found on the helix and are best excised by means of a wedge or V-shaped type of incision. Central lesions are widely excised and amenable to skin grafting of the defect. Posterior lesions lend themselves readily to a rotated skin flap. A margin of 1 cm. should afford uniformly successful results, although follow-up in this group is difficult because of advanced age of the patients. Local recurrence may necessitate sacrifice of the entire auricle. Advanced cancer of the auricle requires sacrifice of the entire pinna and adjacent temporal bone including mastoid. Almost half of the operated cases in this group (13 cases) are free of disease for periods up to five years. The three irradiated cases were failures.

Early lesions of the auditory canal were treated by a sleeve resection of the auditory canal with a replacement of the canal with a split-thickness skin graft maintained in place by a stent. All of the cases (seven) are free of disease for periods up to five years. Advanced cancer of the auditory canal with invasion of the middle ear and mastoid responded best to radical surgical attack. Subtotal resection of the temporal

bone was the surgical procedure of choice. Eight of 31 operated cases are free of disease for periods up to five years; seven cases (23 per cent) are three year cures. The four irradiated cases were failures.

Cancer of the middle ear and mastoid requires temporal bone resection which was utilized in 13 operated cases. Four of these (31 per cent) are three year cures. The four irradiated cases were failures. Combining advanced ear canal and middle ear and mastoid operated cases, (44 cases) the three year salvage is 25 per cent.

Temporal bone resection is a unique combination of intracranial and extracranial surgical *en bloc* resection of cancer-ridden bone. The initial exposure of the dura and petrous pyramid is through a temporal craniotomy. The path of least resistance for cancer extending from the middle ear is through the thin roof of the tympanum into the middle cranial fossa. Once dura and petrous bone are exposed, it becomes at once apparent to the operator whether the disease is limited to the temporal bone or has extended intracranially and to the base of the skull. At this point a decision may be made whether subtotal resection of the petrous and mastoid is practical or whether the disease is unresectable. Occasionally large segments of involved dura may be resected and replaced with temporal fascia. Removal of about 40 cubic centimeters of cerebrospinal fluid will allow the dura and lateral sinus to be retracted from the petrous, so that a Stryker saw and chisels may be introduced to fracture the involved temporal bone from the base of the skull. Hypotensive agents contribute greatly to the reduction of blood loss, and the blood pressure is kept at about 90 systolic throughout the procedure. Fracture of the petrosa is made lateral to the carotid canal and internal auditory meatus. The use of a split thickness skin graft over the operative defect permits rapid healing and reduces the danger of meningitis. Equilibrium is lost for a period of five to 20 days after surgery due to sacrifice of the labyrinth, and lateral tarsorrhaphy compensates somewhat for the facial paralysis.

Thirty-four cases of cancer involving the temporal bone

were subjected to this procedure. Fourteen of these are free of disease for periods of six months to five years. Eleven of the 34 have survived for three years or more (32 per cent three year salvage) and three have survived more than five years. Thirteen cases appeared to be primary in the middle ear and four of these (31 per cent) are three year cures. Four cases (12 per cent) of the 34 died of postoperative cerebral hemorrhage or infection of the brain and meninges.

SUMMARY.

1. Considering the frequency of other affections of the ear, cancer occurs extremely rarely.

2. The auricle most often gives rise to basal cell carcinoma. Squamous cell carcinoma more frequently arises from the auditory canal and middle ear and may metastasize to regional lymph nodes by a rich lymphatic network. Other rare tumors have been classified in this series and are of such rarity as to be curiosities.

3. Because of the difference in therapeutic approach, cancer of the external ear has been classified as being early and advanced, ear canal as early and advanced, and middle ear and mastoid as a therapeutic entity. The surgical approach for small lesions of the pinna involves some sacrifice of the cosmetic appearance, but is remarkably successful. Small lesions originating in the membranous canal are best removed by a sleeve resection with primary skin grafting. Advanced lesions of the auricle, ear canal and middle ear where temporal bone is involved require radical surgery. The most promising procedure for these lesions in the author's experience is a combined intracranial, extracranial subtotal resection of the temporal bone *en bloc*. This operation, while hazardous, upholds the principles of cancer surgery in that cancer-laden bone with a wide margin of normal tissue is removed in a single segment. The three year salvage rate for this type of procedure is approximately 32 per cent.

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CARCINOMA OF SALIVARY GLAND ORIGIN.

A Follow-Up Survey.*

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Eighty patients with microscopically proven carcinoma of salivary gland origin arising in the principal salivary glands or accessory salivary gland tissue have received definitive treatment at the Lahey Clinic during the 15-year period from January 1, 1942, through December 31, 1956. The neoplasm was situated in the parotid gland in 60 patients, the submaxillary gland in 11 patients, the sublingual glands in two patients, and in seven patients the malignant lesion arose in accessory salivary gland tissue located in the lip, cheek, palate, tongue, or floor of the mouth. Follow-up information was obtained in every case and, in each instance, at a date later in 1959 than that of the year during which the patient's treatment was first instituted during the 15-year period. Thus, for those patients in this series whose treatment was initiated most recently the follow-up period was three years.

PATHOLOGY.

Nine pathological diagnoses were made of the malignant neoplastic disease found in this group of patients. These diagnoses have been divided into five groups in order to demonstrate the frequency with which a carcinoma of a particular classification affected the salivary glands in this series (see Table I).

Adenocarcinoma was the tumor most frequently encountered. A pathological diagnosis of adenocarcinoma or one of its subclassifications—carcinoma simplex, papillary carcinoma, cylindroma—was made of the pathological material

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submitted to the pathologists in 54 (67.5 per cent) of the cases. Twenty-nine of these patients are alive, one with recent local recurrence, 17 died of the disease and eight died of disease not related to the neoplasm. The data obtained from an analysis of the patients exhibiting carcinoma of this classification in this series suggests that adenocarcinoma of salivary gland origin is a relatively slow-growing malignant neoplasm with a progressive tendency for local recurrence and a definite trend toward spread to the regional lymph nodes and multiple distant metastases to lung and bone if not completely eliminated by the initial treatment employed. This neoplasm accounted for 41 (73 per cent) of the total number of recur-

TABLE I.

Carcinoma of the Salivary Glands—A Follow-Up Survey—80 Cases. Classification of Tumors and Frequency of Occurrence According to Salivary Gland Involved.

	Parotid	Submaxillary	Sublingual	Accessory Salivary
Malignant mixed tumor	1	0	0	0
Carcinoma	6	1	1	0
Undifferentiated carcinoma	0	0	0	1
Mucoepidermoid carcinoma	7	2	0	1
Epidermoid carcinoma	5	1	0	0
Adenocarcinoma	29	5	1	5
Carcinoma simplex	9	2	0	0
Papillary carcinoma	1	0	0	0
Cylindroma (malignant)	2	0	0	0
Total	60	11	2	7

rences in the 80 patients in this series and, in addition, exhibited distant metastases in 13 (24 per cent) of the 54 patients afflicted with this tumor.

Mucoepidermoid carcinoma occurred in ten (12.5 per cent) patients in this series. Seven are alive, one with distant metastatic disease, and three are dead of the neoplastic disease. A total of four recurrences occurred among three of the patients, one of whom also exhibits the metastases previously mentioned. In addition, generalized metastatic disease developed in one patient and pulmonary metastases in another. Both of these patients are dead. It has been my impression

that mucoepidermoid carcinoma of the salivary glands is a neoplasm of a relatively low grade of malignancy, but an analysis of the ten cases in this series does not substantiate this impression since recurrent disease as well as distant metastases occurred in 30 per cent of the patients.

A diagnosis of malignant mixed tumor was made in only one instance. Carcinoma of an unspecified nature was diagnosed in eight additional patients, however, and these have been included in this classification of malignant mixed tumor. This has been done because Foote and Frazell,* in their discussion of this tumor in their excellent monograph, state that the major portion of such a tumor is malignant, and an appreciable number of these do not exhibit areas of benign mixed tumor when routinely sectioned and, consequently, are diagnosed as carcinoma. Thus, nine patients (11.25 per cent) are included in this classification in this series of 80 patients. Five of these patients are alive and four are dead, one of disease of an unrelated nature. The tumor recurred on six occasions in five of the patients, and three of these exhibited distant metastatic disease at the time of their death.

Epidermoid carcinoma was encountered in only six patients (7.5 per cent); percentagewise, however, it caused more deaths than any other neoplasm in this series. Half of the patients were dead of malignant disease within one year and three months after treatment; furthermore, only one of the three patients in whom the neoplasm recurred following their initial treatment at the Lahey Clinic is alive without further development of neoplastic disease, while two of the six patients exhibited distant metastases at the time of their death.

Undifferentiated carcinoma occurred in only one patient (1.25 per cent) in this series. This patient is alive and free of disease and has had no recurrence. It is impossible to arrive at any conclusion regarding the degree of malignancy of this tumor in view of the fact that it was encountered in only one instance.

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*Foote, F. W., Jr., and Frazell, E. L.: Tumors of the Major Salivary Glands. "Atlas of Tumor Pathology," Section IV, Fascicle 11, 149 pp. Armed Forces Institute of Pathology, Washington, D. C., 1954.

OVER-ALL RESULTS.

Analysis of the data obtained from this survey discloses that 27 of the 80 patients have died of the neoplastic disease for which they received treatment, while 42 are alive and without evidence of recurrent or metastatic disease. Two patients are alive with disease, one of whom exhibits a local recurrence and the other multiple distant metastases. Nine other patients have died of unrelated disease without recurrence of or metastases from the original neoplasm. In addition, a total of 55 recurrences developed at one time or another in 37 of the 80 patients comprising this series (see Table II).

TABLE II.

Over-All Results.

	Cases	Per Cent
Dead—		
Of neoplasm	27	33.7
Of other disease	9	11.3
Alive—		
With neoplasm	2	2.5
Without neoplasm	42	52.5
Total	80	
Recurrence	37	

Of the patients whose deaths were attributed to other diseases, seven were treated for carcinoma of the parotid gland and one each for carcinoma of the submaxillary gland and an accessory salivary gland. All but two of the patients had survived for three to nine years without evidence of recurrent malignant disease. The remaining two patients died within five months after surgical excision of the neoplasm (see Table III).

When the data derived from this study are considered in terms of the salivary gland involved by the malignant process, it is seen that malignant neoplasms of the submaxillary gland are associated with a higher death rate than those of the parotid, sublingual or accessory salivary glands. Seven of the patients with carcinoma of the submaxillary gland died

of the disease, while only three are alive without disease. One patient died of unrelated disease.

Although carcinoma of the parotid gland occurred more than five times as frequently as that of the submaxillary gland, only 18 patients died of the disease, while 33 are alive and clinically free of recurrence or metastases. Seven of the 60 patients in this group have died of unrelated disease and two are alive with persistent disease, one with local recurrence of the neoplasm and the other with generalized metastases.

TABLE III.

Dead of Other Disease (No Recurrence).

Cause of Death	Interval After Operation Years	Months	Days
Parotid Gland			
Cerebral thrombosis	9	9	19
Acute myocardial infarction	9	8	21
Coronary thrombosis	7	1	5
Bronchopneumonia	6	3	10
Cancer of stomach	4	3	3
Papillary carcinoma of thyroid	3	1	10
Congestive heart failure	0	4	21
Submaxillary Gland			
Coronary thrombosis	0	5	0
Accessory Salivary Glands			
Cirrhosis of liver	3	2	23

Malignant tumors of aberrant tissue origin are generally considered to carry a graver prognosis than do those arising from normally placed tissue. Tumors of salivary gland origin which are situated in the tongue, cheek, lip, palate or floor of the mouth are often referred to as being of aberrant salivary gland origin. This is a misnomer, since these tumors do not arise from aberrant structures but from normally situated accessory salivary glands which are constant anatomical components of the mucous membrane of these regions. In seven patients in this series a malignant neoplasm of salivary gland origin was located either in the lip, cheek, palate, tongue or floor of the mouth. Five are alive without evidence of persistent disease, and two are dead. One of these died of the carcinoma and the other died of cirrhosis of the liver.

In two cases the carcinoma originated in the sublingual glands. One of these patients is dead of the neoplastic disease, and the other is alive without evidence of disease (see Table IV).

FIVE-YEAR RESULTS.

Sixty-seven patients have had follow-up studies for at least five years from the date of initial treatment at the Lahey Clinic, for carcinoma of salivary gland origin. The tumor was situated in the parotid gland in 49 patients, the sub-

TABLE IV.

Total Results According to Gland Involved.

	Parotid Cases	%	Submaxillary Cases	%	Sublingual Cases	%	Accessory Salivary Cases	%
Dead—								
Of neoplasm	18	— 30	7	— 64	1	— 50	1	— 14.3
Of other disease	7	— 12	1	— 9	0		1	— 14.3
Alive—								
With neoplasm	2	— 3	0		0		0	
Without neoplasm	33	— 55	3	— 27	1	— 50	5	— 71.4

maxillary gland in ten, the sublingual gland in two and in accessory salivary glands in six patients.

Eleven of the patients who had been treated for carcinoma of the parotid gland are dead of the disease. Another died of cancer of the stomach nine months prior to the termination of the five-year period. Thirty patients are alive and have exhibited no evidence of recurrent or metastatic malignant disease for five years or more. Six of these had had recurrence of the carcinoma but following additional treatment have had no further recurrence of the malignant process for at least five years. Twenty-four of the 30 patients have never had recurrent disease. In two other patients recurrences developed before the end of the five-year period and after further therapy they are alive but have been free of malignant disease for less than five years. Five other patients were alive at the termination of the five-year interval but presented either local recurrences or metastatic disease.

Four of the ten patients treated for carcinoma of the submaxillary gland are dead of the disease. Another patient died of coronary thrombosis five months after a local recurrence had been excised. Four patients are alive. In two of these the original neoplastic disease had not recurred during the five-year interval, and in the other two recurrent malignant disease had developed but had been eradicated by additional therapy. Both of these patients have been alive for more than five years but have been free of the disease for less than five years. One patient was alive with disease

TABLE V.

Five-Year Follow-Up Results According to Gland Involved. 67 Cases.

	Parotid Cases	%	Submaxillary Cases	%	Sublingual Cases	%	Accessory Salivary Cases	%
Dead—								
Of neoplasm	11	23	4	40	1	50	1	17
Of other disease	1	2	1	10	0		1	17
Alive, no recurrence	24	49	2	20	1	50	2	33
Recurrence—								
Alive without disease five years after treatment	6	12	0		0		2	33
Alive without disease less than five years	2	4	2	20	0		0	
Alive with neoplasm	5	10	1	10	0		0	

at the end of five years but has since died of pulmonary metastases.

One of two patients with carcinoma of the sublingual gland is dead of the disease, while the other patient is alive without evidence of neoplastic disease.

Of the six patients with carcinoma of the accessory salivary glands, one is dead of the disease, and another died of cirrhosis of the liver without further evidence of malignant disease. Four patients have been alive five years or longer without malignant disease. In two of these recurrent or metastatic disease had never developed after their initial treatment at the clinic. In the other two patients additional neoplastic disease had developed, one a local recurrence and the other metastases to the lymph nodes of the submaxillary triangle

from a neoplasm of the upper lip. Both of these responded satisfactorily to surgical excision (see Table V).

TEN-YEAR RESULTS.

Thirty patients in this survey have had follow-up studies for at least ten years from the date of initial treatment for neoplastic disease at the Lahey Clinic. The tumor was located in the parotid gland in 22 patients, the submaxillary gland in five, the sublingual glands in one and an accessory salivary gland in two.

Of those patients who had been treated for carcinoma of the parotid gland, 11 died of the disease and one of unrelated disease. The latter patient had had no recurrence of the original primary adenocarcinoma and had died of cerebral thrombosis nine years and ten months after excision of the neoplasm. Five patients who are alive have had no recurrence of the neoplastic process during this period. Recurrence had developed in four patients and all four have been alive for at least ten years but not free of disease during the ten-year period. One of these patients has since died of acute myocardial infarction just three months short of ten years' freedom from the disease. The remaining patient is alive but presents local recurrence of the carcinoma.

Only one of the five patients with carcinoma of the submaxillary gland had no recurrence of the tumor and was alive and clinically free of neoplastic disease ten years or more after initial treatment at the Lahey Clinic. Another patient has been alive ten years, but the tumor had recurred which necessitated further treatment and, thus, has not been free of malignant disease for ten years. The remaining three patients died of carcinoma of the submaxillary gland.

Only one patient with carcinoma of the sublingual glands has had follow-up studies for ten years or longer; a recurrence has never developed in this case.

Two patients with carcinoma of accessory salivary glands are included in this group. One of these had a recurrent neoplasm when originally treated at the Lahey Clinic, and

this patient died of cerebral infarction one week after excision of the lesion. This patient has been included among those who died of malignant disease in the over-all statistical results. The other patient had had metastases to the submaxillary triangle from an adenocarcinoma of the upper lip and has had no further recurrence for more than five years (see Table VI).

TABLE VI.

Ten-Year Follow-Up Results According to Gland Involved. 20 Cases.

	Parotid Cases	%	Submaxillary Cases	%	Sublingual Cases	%	Accessory Salivary Cases	%
Dead—								
Of neoplasm	11	— 50.0	3	— 60	0		1	— 50
Of other disease	1	— 4.5	0		0		0	
Alive, no recurrence	5	— 22.7	1	— 20	1	—100	0	
Recurrence—								
Alive without disease ten years after treatment	0		0		0		0	
Alive without disease less than ten years	4	— 18.1	1	— 20	0		1	— 50
Alive with neoplasm	1	— 4.5	0		0		0	

RECURRENCE.

Forty-six of the patients in this series had had no treatment before coming to the Lahey Clinic, while 34 patients had received treatment elsewhere and exhibited recurrent carcinoma of salivary gland origin. In 34 of the 46 patients who had had no previous treatment the neoplasm was located in the parotid gland, six patients had a malignant tumor of the submaxillary gland, one a malignant tumor of the sublingual gland and, in the remaining five patients, the malignant neoplasm originated in accessory salivary gland tissue. The 34 recurrent tumors involved the parotid gland in 26 cases, the submaxillary gland in five, the sublingual gland in one and the accessory salivary glands in two patients (see Table VII).

A recurrence of the carcinoma, either local or metastatic to the regional lymph nodes, developed in 17 (approximately 37 per cent) of the 46 patients who received their initial treatment at this clinic—in the parotid gland in 11 cases, in the

submaxillary gland in four, and in accessory salivary glands in two. In contrast, further recurrent disease developed in 20 (approximately 59 per cent) of the 34 patients who were first treated at the Lahey Clinic for recurrent carcinoma of the salivary glands. The additional recurrent neoplastic disease was situated in the parotid gland in 17 patients, the submaxillary gland in two and the sublingual gland in the remaining patient (see Table VIII).

TABLE VII.

Status of Malignant Neoplasm at Initial Examination.

Gland	Neoplasm	
	Primary	Recurrent
Parotid	34	26
Submaxillary	6	5
Sublingual	1	1
Accessory salivary	5	2
Total	46	34
	80	

TABLE VIII.

Patients Exhibiting Recurrence After Treatment at Lahey Clinic.

Gland	Neoplasm			
	Primary		Recurrent	
	Cases	Per Cent	Cases	Per Cent
Parotid	11	32	17	65
Submaxillary	4	67	2	40
Sublingual	0		1	100
Accessory salivary	2	40	0	
Total	17		20	
	37			

These 37 patients in whom recurrent carcinoma developed presented a total of 55 recurrences. Forty-two of these occurred in the parotid gland, but only 15 (27 per cent) occurred in those patients whose initial treatment was for primary carcinoma as compared to 27 (49 per cent) which developed in patients whose original treatment was for recurrent disease. In three of the patients who had been treated initially for primary carcinoma more than one recurrence developed,

while four of those patients who had received treatment for recurrent disease exhibited more than one additional recurrence. A total of ten recurrences developed in those patients treated for carcinoma of the submaxillary gland and, in contradistinction to the parotid gland, eight (80 per cent) of these occurred in patients treated initially for primary carcinoma while only two (20 per cent) developed in patients whose original treatment was for recurrent malignant disease. An additional recurrence developed on one occasion in a patient who had received treatment for a recurrent carcinoma of the sublingual gland and a recurrence developed in each of two patients treated for carcinoma of the accessory salivary glands (see Table IX).

TABLE IX.

Total Number of Recurrences After Treatment at Lahey Clinic.

Gland	Neoplasm	
	Primary	Recurrent
Parotid	15	27
Submaxillary	8	2
Sublingual	0	1
Accessory salivary	2	0
Total	25	30

Only 15 of these 37 patients in whom recurrence of the neoplastic process developed after their first treatment at the Lahey Clinic are alive in spite of additional therapy for the recurrence. Thirteen are alive and free of malignant disease while the other two are alive but exhibit either local recurrent or metastatic malignant disease. Twenty-two of the 37 patients are dead, two of unrelated disease and 20 of the malignant tumor (see Table X).

METASTASES.

Metastases or extension of the malignant process to structures other than the regional lymph nodes occurred in 21 patients (26.3 per cent) in this series and only in those patients with carcinoma of the parotid or submaxillary glands. No apparent relationship of a significant degree appeared to exist between a pathological diagnosis and the incidence of

metastatic disease percentagewise; however, there was a definite relationship between the incidence with which metastases occurred and whether or not the neoplasm had recurred locally. Only three of the patients in whom distant metastases developed had not had a local recurrence of the neoplastic disease, and in one of these the original neoplasm had not resolved as a result of the therapy employed.

TABLE X.

Status of 37 Patients Treated for Recurrence of Neoplasm Following Initial Treatment at Lahey Clinic.

	Cases	Per Cent
Alive—		
Without further recurrence	13	35.1
With persistent or metastatic neoplasm	2	5.4
Dead—		
Of neoplasm	20	54.1
Of other disease	2	5.4

TABLE XI.

Metastases or Extension to Structures Other Than Regional Lymph Nodes.

Gland	Neoplasm			
	Primary		Recurrent	
	Cases	Per Cent	Cases	Per Cent
Parotid	8	24	7	27
Submaxillary	2	33	4	80
Sublingual	0		0	
Accessory salivary	0		0	
Total	10		11	
	21			

Of 15 patients (25 per cent) with carcinoma of the parotid gland in whom distant metastases occurred, eight had had no treatment for the malignant disease prior to that instituted at the Lahey Clinic (see Table XI). Thirteen of the 15 patients had had local recurrence of their disease, while two patients who had had no treatment before that instituted at the Lahey Clinic had not developed recurrent disease. In one of these two, recurrence had not developed at any time before the metastatic disease was discovered; in the other patient the primary carcinoma had never responded satisfactorily to treatment. Distant metastatic disease developed in six (54.5 per

cent) of 11 patients with carcinoma of the submaxillary gland, and only one of these patients had not had a local recurrence of the malignant tumor. The malignant tumor in one patient was recurrent when the patient was first seen, and treatment of this lesion proved unsuccessful.

Pulmonary metastases occurred most commonly in this series, there being nine instances of metastases to the lungs. Metastatic disease involving bone was next in frequency with six such metastatic lesions. Metastases or direct extension to the brain occurred on three occasions and to the scalp and liver in one instance each. The case of metastasis to the liver is of particular interest as it occurred 16 years after excision

TABLE XII.

Location of Metastases.

Lungs		9
Bone		6
	Ribs	2
	Pelvis	2
	Skull	1
	Spine	1
Scalp		1
Liver		1
Brain		3
Generalized		5

of a recurrent carcinoma of the submaxillary gland, without further local recurrence. Five patients exhibited generalized metastatic disease (see Table XII).

SUMMARY AND CONCLUSIONS.

Eighty patients have been treated at the Lahey Clinic for carcinoma of salivary gland origin during the 15-year period from January 1, 1942, through December 31, 1956. Follow-up information regarding the patient's status in 1959 has been obtained in every instance and has been correlated insofar as possible with the day and month of the year the patient first received definitive treatment at the Lahey Clinic. Thus, every patient who is now alive has had follow-up studies for three years or longer.

Forty-four patients, two of whom exhibited either persistent or metastatic disease, were alive at the time this follow-up survey was completed—a survival rate of 55 per cent. Thirty-six patients were dead, 27 of the malignant neoplastic disease for which they had been treated and the remaining nine patients of other disease, without evidence of recurrent or metastatic disease.

On the basis of this survey, carcinoma of the submaxillary gland must be considered to have much greater lethal potentiality than carcinoma of the parotid gland. Sixty-four per cent of those patients with carcinoma of the submaxillary gland died of the neoplastic process as compared to 30 per cent of the patients with carcinoma of the parotid gland. In addition, the incidence of distant metastases was higher in carcinoma of the submaxillary gland than carcinoma of the parotid gland. Distant metastatic disease ultimately developed in six of the 11 patients (54.5 per cent) with carcinoma of the submaxillary gland, as compared to 15 (25 per cent) of the 60 patients with carcinoma of the parotid gland who exhibited such metastases. The increased incidence of distant metastases with carcinoma of the submaxillary gland apparently was not related to the type of neoplasm diagnosed in this series since there was no significant difference in this incidence among the various tumor classifications. Rather, the incidence of metastases appeared to be related to the incidence of recurrent disease since the percentage of recurrent disease was greater with carcinoma of the submaxillary gland than with carcinoma of the parotid gland.

The malignant neoplasm recurred much more frequently among those patients whose initial treatment at the Lahey Clinic was for recurrent carcinoma. Further recurrence developed in 59 per cent of these patients as compared with a recurrence rate of 37 per cent for the patients initially treated for primary carcinoma of the salivary glands.

Recurrence developed in 11 of 34 patients who received treatment for primary carcinoma of the parotid gland, whereas additional recurrent disease developed in 17 of 26 patients who had recurrent carcinoma of the parotid gland when first

treated at the Lahey Clinic. Only three of the 11 patients who had recurrent disease after treatment of their primary carcinoma are alive and free of disease at this time—a salvage rate of 27 per cent. Six of the 17 patients in whom additional recurrence developed after treatment for their original recurrent neoplastic disease are alive and free of disease at this time—a salvage rate of 35 per cent.

Recurrent disease developed in four of the six patients who were treated for primary carcinoma of the submaxillary gland. Only one patient is now alive and free of disease. Further recurrent disease developed in two of five patients who received treatment for recurrent carcinoma of the submaxillary gland when first seen at the Lahey Clinic. Only one of these patients is alive without disease at this time. Thus only two of the six patients treated for recurrent malignant disease of the submaxillary gland are now alive and free of disease. One patient who was treated for a recurrent carcinoma of the sublingual gland had further recurrence and is now dead of malignant disease. Two other patients who were treated for primary carcinoma of the accessory salivary glands and in whom the neoplasm recurred are now alive and free of disease.

Thus, of the 37 patients who had recurrent malignant disease following their original treatment for carcinoma of salivary gland origin during the 15-year period from January 1, 1942, through December 31, 1956, only 13 patients are now alive and free of the malignant process—a survival rate of 35 per cent.

Since all but nine of the carcinomas in this series were situated in the parotid or submaxillary glands, it would seem that the survival rate should be higher than that obtained in this series. Tumors located in these situations are recognizable as such early in their course and are readily accessible to adequate therapy; thus, it would seem that the survival rate of 57.5 per cent for primary carcinomas of these glands obtained in this series could be increased if adequate treatment were instituted early in the course of the disease.

CARCINOMA OF THE TONSIL.*

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Cancer of the tonsil accounts for 1.5 to 3 per cent of all cancers of the body. This figure, however, does not indicate the magnitude or importance of the problem for the otolaryngologist whose patients represent a selected population having symptoms in the tonsillar region. There are two parts to the clinical problem of cancer of the tonsil: first is the diagnosis of the early or occult lesion. Because the area is relatively insensitive, early symptoms of cancer of the tonsil are vague and indefinite. Soreness or a sticking sensation on one side of the throat, otalgia or fullness in the upper neck may be the first indication of a new growth. When these symptoms occur in a male past 50 years of age, they are of particular significance because of the higher incidence of cancer of the tonsil in this group. Slight changes in the appearance of the tonsil, redness about a crypt, or a thickened area which bleeds easily should be regarded with suspicion and biopsy specimens taken. Lesions diagnosed at this early stage are curable in a high percentage of cases.

The second part of the problem of cancer of the tonsil is the extensive lesion which presents no difficulty in diagnosis but is a challenge in management. This is a responsibility shared by the otolaryngologist and radiation therapist. Treatment should be based on an accurate estimate of the extent of the disease which can be best accomplished by staging the tumor. This report covers our experience in attempting to stage a series of 30 cases of cancer of the tonsil.

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ANATOMY.

The anatomy of the tonsillar region is familiar to every otolaryngologist. The tonsillar bed was his cradle and on its pillars he cut his first surgical teeth. The glosso-palatine fold, the pharyngo-palatine fold, better known to us as the anterior and posterior pillars, the upper and lower poles and the superior constrictor muscle of the pharynx, are its boundaries. This is the anatomical bed for the tonsil; however, cancer does not respect precise anatomical boundaries; it spreads to the adjacent structures where it may grow in bulk, at times even larger than at the site of origin. In some lesions it may be difficult or impossible to determine whether the tumor started in the tonsil and spread to the anterior pillar, tongue or palate, or whether it had its origin in one of these sites and later involved the tonsil; however, in most instances the site of origin can be determined. The importance of classifying tumors according to site of origin is based on the observation that tumors arising in different sites appear to be biologically different; they behave differently and respond to therapy differently. Cancer of the tonsil is biologically different from cancer arising in the anterior pillar, and each should be considered as a separate clinical entity. The former tends to be more radio-sensitive; the latter is more radio-resistant. The 30 cases in this report are all cancers which arose primarily in the tonsil.

The tonsil has a rich blood supply and a well developed lymphatic drainage; this, plus the constant muscular activity of the area provides conditions conducive to early metastasis. Undoubtedly, these factors account in part, for the 70 to 80 per cent of clinically palpable nodes when the patient is first seen.

PATHOLOGY.

Tumors arise either from the epithelium covering the tonsil or from the lymphatic tissue. Consistent with many reported series, 80 per cent of the 30 tumors are of epithelial origin. Three others were lymphoepithelioma and three were lympho-

sarcoma. We have accepted the method of reporting which groups lymphoepithelioma with lymphosarcoma.

STAGING OF CANCER OF THE TONSIL.

The necessity for staging in the clinical study of cancer has had widespread acceptance throughout the medical world, and its importance is indicated by the number of national and international committees that are working toward forming acceptable criteria for staging. Behind this movement is the basic conviction that reports of cure rate for any form of

TABLE I.

Carcinoma of the Tonsillar Area.
Survival rates reported in literature, showing spread of cure rates.

Author	Period	No. of Cases	Per Cent Survived Five Years
1. Berven	1925-1945	107	16.2
2. Cade and Ledlie	1939	82	21
3. Coutard	1920-1926	33	18
4. Duffy	1921-1928	49	20
5. Ennuyer and Bataini	1919-1954	534	18
6. Jacox	1930-1958	65	32
7. Martin and Sugarbaker	1930-1935	107	18
8. Parshall and Stenstrom	1926-1950	72	21
9. Paterson	1940-1944	52	12
10. Scanlon, et al.	1958	46	43
11. Sheline, et al.	1931-1950	56	19
12. Teloh	1952	142	5
13. Walker and Schulz	1936-1945	39	15
14. Wood and Aird	1934-1945	80	29

therapy are not meaningful unless the stage of the lesion has been considered.

The advantages of staging are many. It would avoid widespread disparity in reported cure rates of cancer of the tonsil which may be due in part to the difference in distribution of early, moderately advanced and advanced lesions in each series (see Table I); it provides a more accurate basis for comparing results from different institutions; it makes it possible to summate the experience of many clinics; furthermore, the very process of staging disciplines the clinician to make a more thorough investigation of each lesion. The fruit

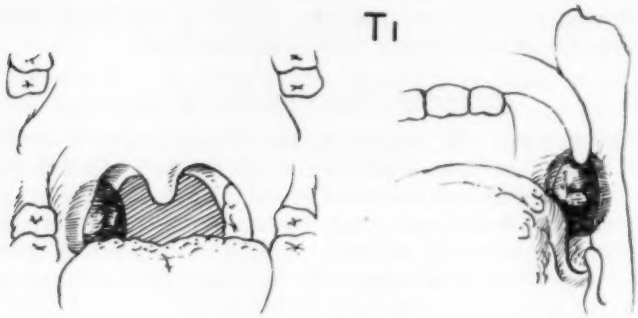


Fig. 1-a. T1. Confined to the tonsillar bed and less than 3 cm. diameter. Exophytic and bulky type.

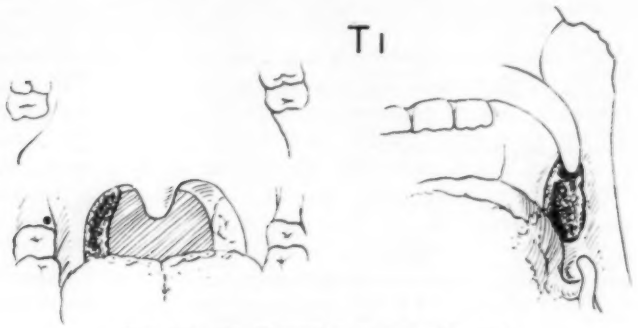


Fig. 1-b. T1. Ulcerative or granular, flat.

of such efforts is a more sound clinical judgment with respect to treatment and prognosis.

DESCRIPTION OF STAGING OF THE PRIMARY TUMOR.

We have undertaken to stage cancer of the tonsil using the system proposed by the International Union Against Cancer, which is generally referred to as the T.N.M. system.

T1. Confined to the tonsillar bed and less than 3 cm. diameter (see Fig. 1).

a. Exophytic, bulky.

b. Ulcerative or granular, flat.

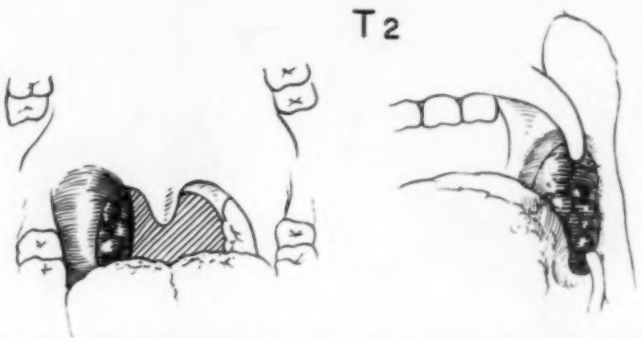


Fig. 2-a. T2. Minimal extension to adjacent structures; approximately 3 to 5 cm. diameter. Exophytic, bulky tumor, with predominant extension to hypopharynx.

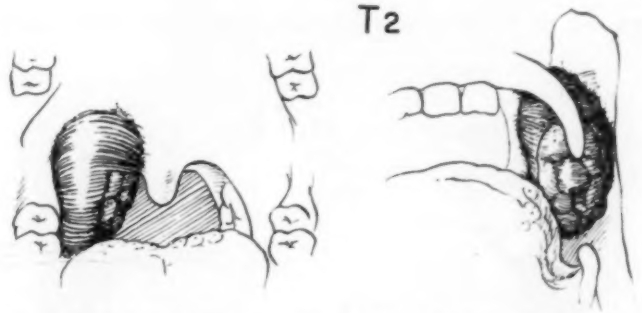


Fig. 2-b. T2. Exophytic, bulky tumor with predominant extension to palate and toward the nasopharynx.

T2. Minimal extension to adjacent structures: approximately 3 to 5 cm. diameter (see Fig. 2).

a. Exophytic, bulky with extension to hypopharynx.

b. Exophytic, bulky with extension to palate and toward the nasopharynx.

c. Ulcerative, flat, with extension into the glossotonsillar sulcus.

d. Ulcerative, flat, with extension into the tonsillar pillars.

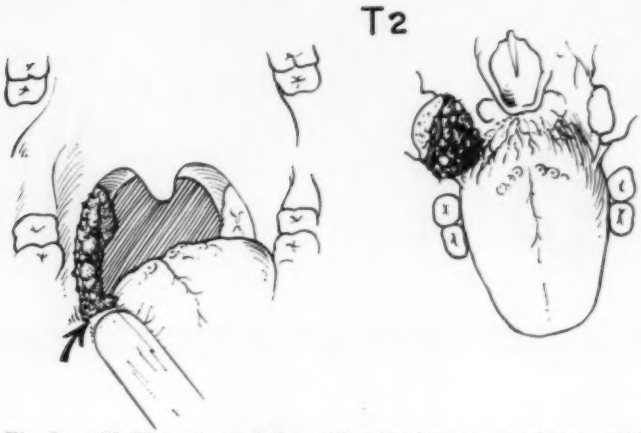


Fig. 2-c. T2. Ulcerative and flat, with extension into the glosso-tonsillar sulcus.

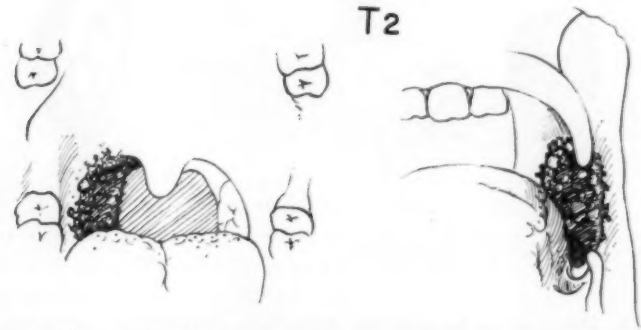


Fig. 2-d. T2. Ulcerative and flat, with extension into the tonsillar pillars.

T3. Large or extensive lesion with moderate infiltration into surrounding structures; larger than 5 cm. (see Fig. 3).

a. Exophytic, bulky with extension into nasopharynx and hypopharynx, and forward displacement of the soft palate.

b. Bulky tumor with submucosal extension into the palate.

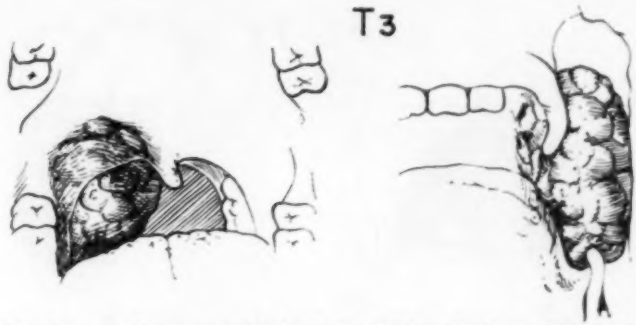


Fig. 3-a. T3. Large or extensive lesion with moderate infiltration into surrounding structures; larger than 5 cm. Exoyhytic bulky tumor, extending into nasopharynx and hypopharynx, and with forward displacement of the soft palate.

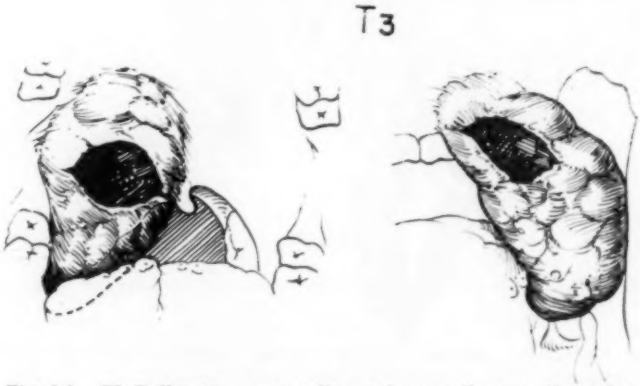


Fig. 3-b. T3. Bulky tumor extending submucosally into the palate.

c. Massive tumor with narrow base and moderately infiltrating adjacent structures.

d. Ulcerative lesion with moderate invasion of adjacent structures.

T4. Massive lesion (see Fig. 4).

a. Excavating necrotizing lesion with large crater.

b. Extensive infiltration of tongue with deep linear fissure.

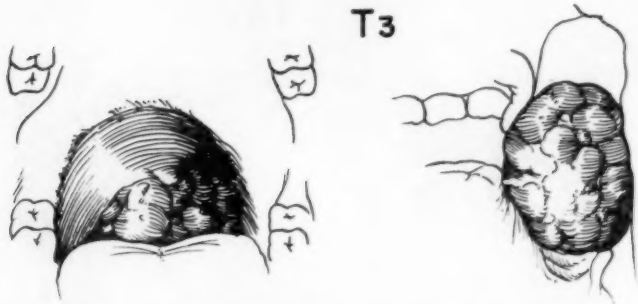


Fig. 3-c. T3. Massive tumor, but with moderate infiltration of adjacent structures at the base of the tumor.

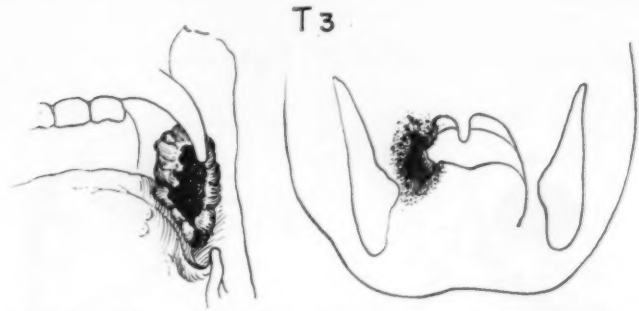


Fig. 3-d. T3. Ulcerative lesion with moderate invasion of adjacent structures.

c. Extension across faucial arch; involvement of both tonsils.

DESCRIPTION OF METASTATIC CERVICAL NODES.

Cervical nodes are classified into four categories from NO to N3 as follows:

NO—No node.

N1—Single small to moderate size (less than 3 cm.) or two adjacent small nodes (less than 2 cm. each).

N2—Large, moveable node (more than 3 cm.) or multiple unilateral nodes (more than 2 cm.).

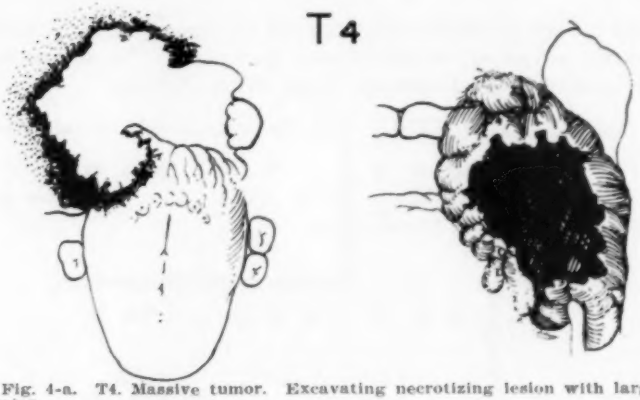


Fig. 4-a. T4. Massive tumor. Excavating necrotizing lesion with large crater.

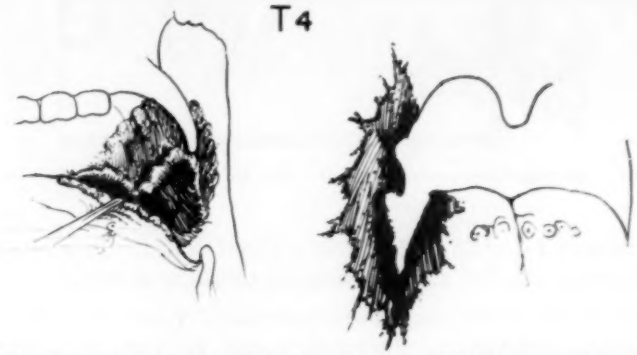


Fig. 4-b. T4. Extensive infiltration of tongue with deep linear fissure.

N3—Fixed large unilateral node or nodes, or bilateral nodes.

DESCRIPTION OF DISTANT METASTASIS.

Distant metastasis is designated as **M**, followed by a description of the site.

DESCRIPTION OF STAGES OF CANCER OF THE TONSIL.

The T.N.M. categories make it possible to describe any malignant lesion. These categories are analyzed and separated

into groups indicating the stage of the lesion as early, moderately advanced, advanced, and incurable. The stages are designated Stage I through Stage IV, as follows:

Stage I—T1NO, T2NO. The cure rate should be relatively high.

Stage II—T1N1, T2N1, T3NO, T3N1. Cure rates are expected in moderate percentages.

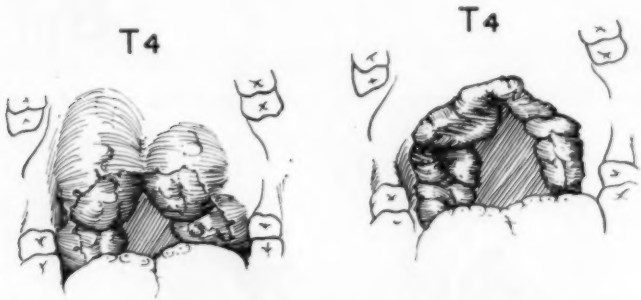


Fig. 4-c. T4. Extension across faucial arch; involvement of both tonsils.

Stage III—T1N2, T2N2, T3N2, T4NO, T4N1. Cure rate is extremely low, but aim of treatment should be a cure.

Stage IV—T4N2 and all combinations of T with N3; also M (distant metastasis). Incurable except for rare unexpected case.

The stages are represented on the T. N. diagram (see Fig. 5) with heavy lines. The distribution of the 30 cases is shown by the black dots.

Classification and staging, expressed in its simplest terms, is an attempt at speaking a common language. An early lesion fits in the definitions of Stage I; a moderately advanced lesion is Stage II; an advanced lesion is Stage III; and far advanced or lesions with a hopeless prognosis are Stage IV. All combinations with M would be considered incurable and are placed in Stage IV.

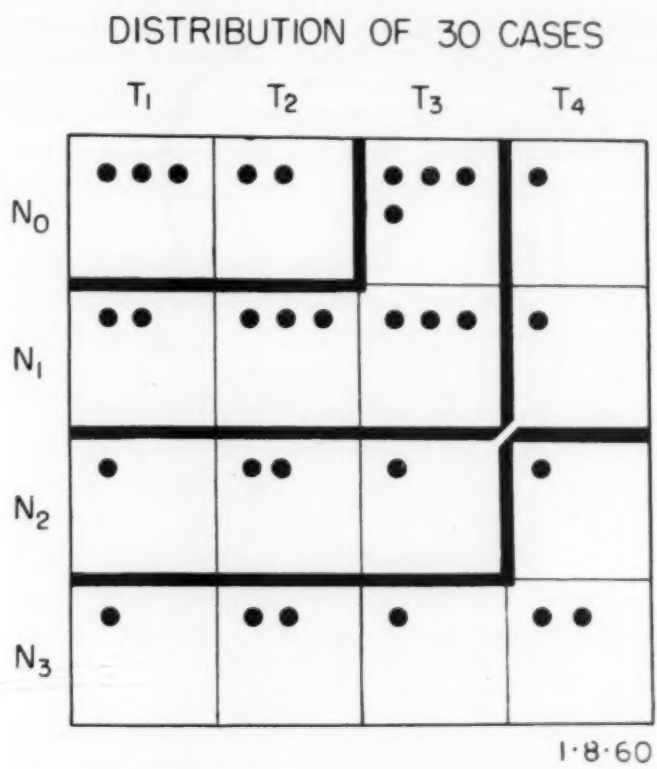


Fig. 5. Distribution of the 30 malignant tumors of the tonsil in this series. It includes 24 epitheliomas, three lymphoepitheliomas and three lymphosarcomas.

TREATMENT.

a. Radiation Therapy:

Supervoltage radiation, either with an X-ray machine or cobalt bomb, has been one of the advances in modern treatment of cancer. New techniques, particularly rotation, have further improved the effectiveness of radiation. All 30 patients in this report have been treated with a two million volt X-ray machine using the rotation technique (see Fig. 6).

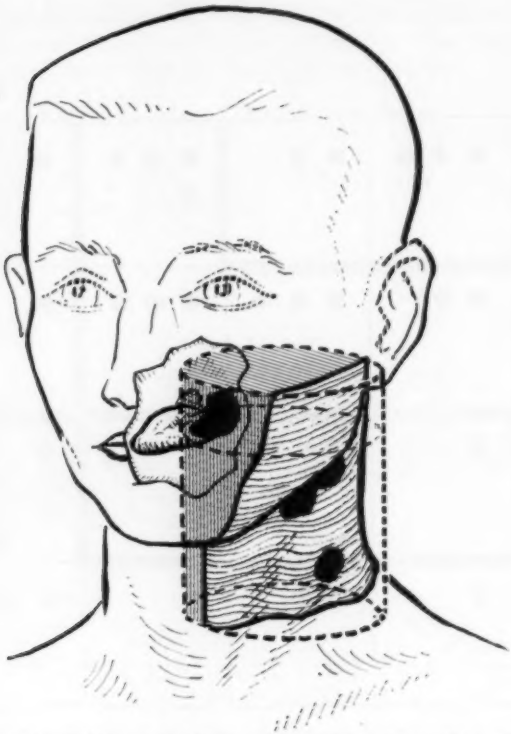


Fig. 6. Distribution of irradiation achieved by supervoltage X-rays and the rotation technique. The partially immersed cylinder of intense irradiation includes the primary tumor and neck nodes "in continuity."

This technique delivers a concentrated dose of radiation in the region of the tumor and the neck nodes with minimal irradiation effect on the adjacent normal tissues. For cancer of the tonsil the shape of the volume of intense irradiation is a cylinder which includes the tonsillar region and the upper neck nodes in continuity. The accuracy of the rotation is checked on an X-ray film inserted in a phanthom of the head, and consequently lends and neck at the level of the lesion.

Cancer of the tonsil is one of the more radio-sensitive cancers of the upper respiratory tract

itself to treatment with external radiation alone. Most lesions can be destroyed with tumor dosage ranging from 5500 to 6500 Roentgens in three to five weeks. An occasional squamous cell carcinoma may be resistant and require a larger dose.

The skin reaction is mild as a result of supervoltage X-rays. Mucous membrane reaction is confined to one side of the

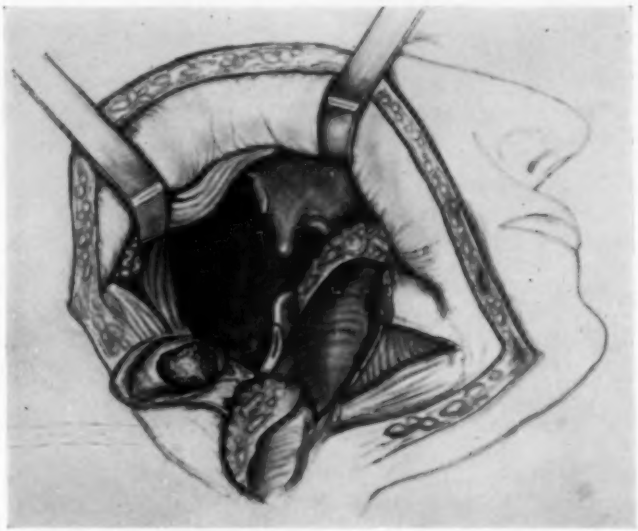


Fig. 7. Surgical resection of tonsillar region (modified from Ennuyer).

pharynx by virtue of the rotation technique; this markedly reduces the discomfort of the patient which, in the past, has often been a limiting factor.

b. Surgery:

Surgery has an important but secondary role to play in the management of cancer of the tonsil. The indications for surgery are three: *1.* failure of radiation therapy to control the primary tumor, or recurrence of the primary lesion, *2.* cervical nodes which are palpable following radiation, par-

ticularly in the more radio-resistant carcinomas and *3.* osteomyelitis of the mandible or non-healing radiation ulceration of the tonsillar region. The sloughing ulceration and osteomyelitis produces intractable pain and dysphagia which makes it impossible for the patient to maintain his nutrition. The complications of radiation therapy can be as fatal as the cancer iteslf. Death may come from a sudden massive hemorrhage. A wide surgical excision relieves the patient of the foul slough and the intractable pain and restores his well-being.

The area excised includes the primary site, portion of the mandible, palate, pharynx, pterygoid muscles, floor of mouth and tongue (see Fig. 7).

Closure is accomplished by using the skin and soft tissues of the neck. The pharyngeal defect is too large for a primary closure and is allowed to granulate in.

It is remarkable that the external closure holds without fistula formation, in spite of the fact that the tissues have been heavily irradiated. Two factors that determine whether the closure will be maintained are: *1.* the period of time elapsing between the completion of radiation and the surgery and *2.* the total amount of radiation given to the area.

If the surgical excision is done within the first year and the radiation tissue dose had been less than 6000 Roentgens with supervoltage X-rays, secondary wound breakdown will be infrequent. The cosmetic and functional results are acceptable (see Figs. 8-a and 8-b).

RESULTS.

Our cases are small in number and have not matured, hence, reporting our experience will not have the weight of a larger series observed for a longer period of time; however, our series has the virtue of presenting a single form of therapy managed by one group. Although final judgments cannot be made concerning the effectiveness of supervoltage rotation technique, a review of our experience at this time is

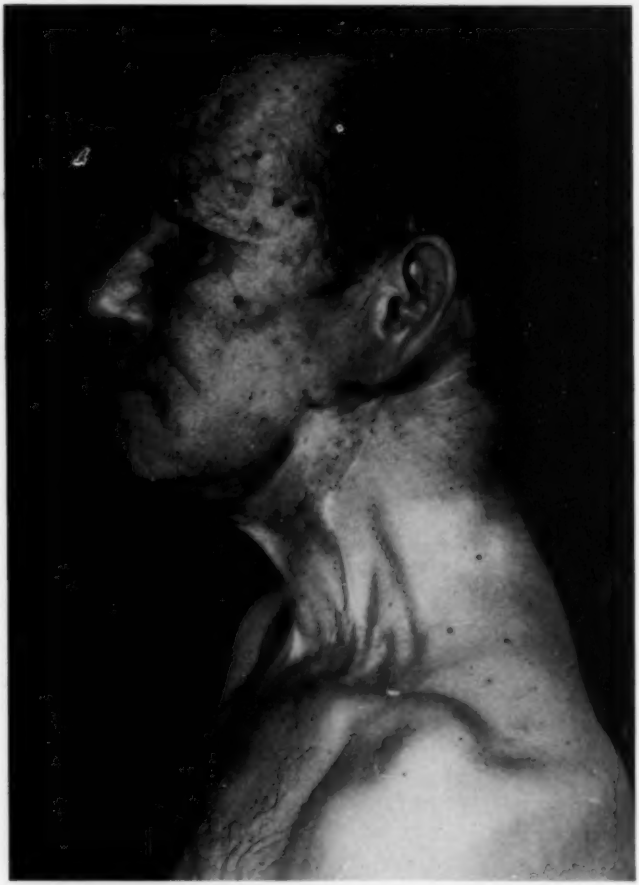


Fig. 8-a. Postoperative photograph (lateral view).

of some value. The course of each patient is recorded on the flow chart (see Fig. 9).

The results among the 22 patients with epithelioma and lymphosarcoma and lymphoepithelioma of the tonsil, treated more than two years ago, were first studied (see Fig. 10). Though the number of cases is too small to be significant,

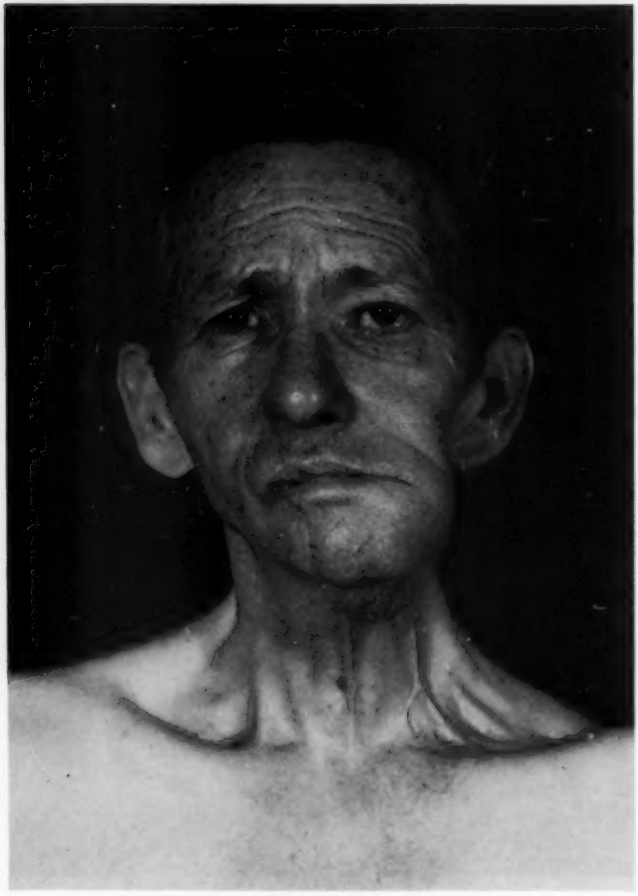


Fig. 8-b. Postoperative photograph (front view).

there appears to be a correlation between stage and prognosis. In the Stage I cases, the two year arrest rate was 100 per cent, Stage II, 66 per cent, Stage III, 33 per cent and in Stage IV none was arrested. In the past it has been assumed that in defining progressive stages of disease, metastatic nodes constituted a more advanced stage than no nodes. Our opin-

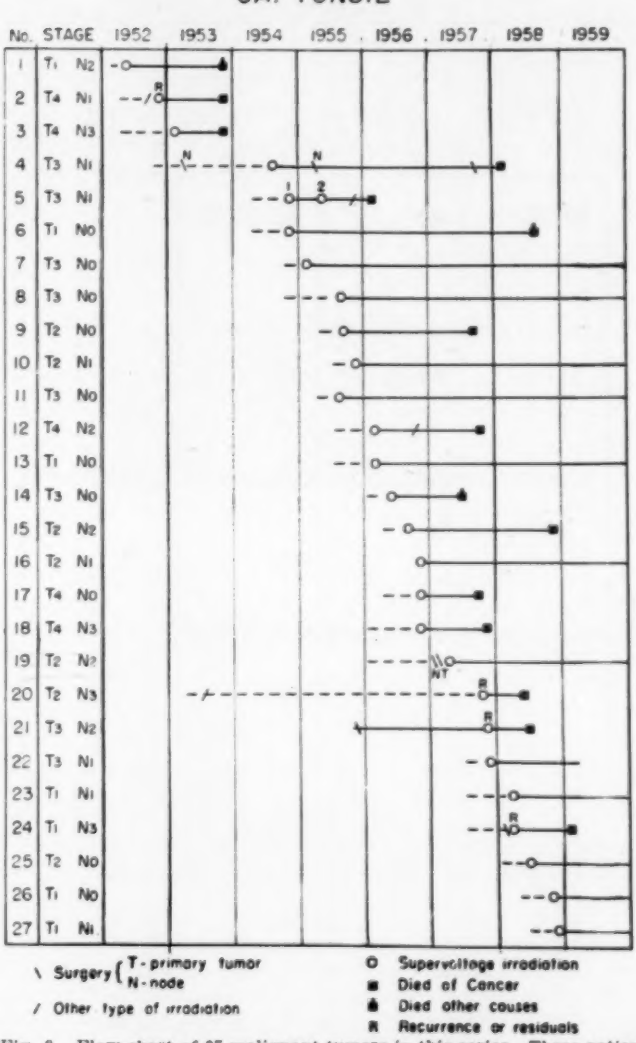
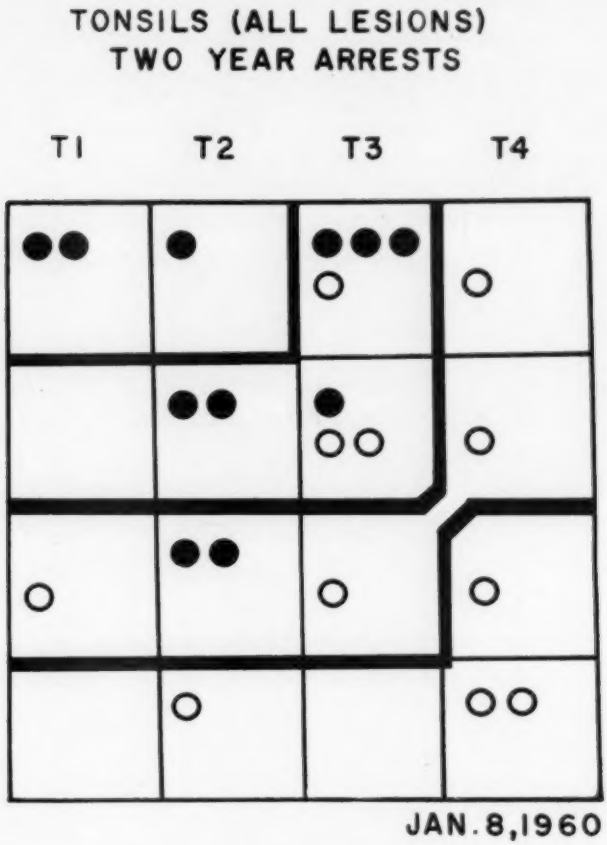


Fig. 9. Flow chart of 27 malignant tumors in this series. Three patients treated less than one year ago are not depicted. The dotted line represents duration of symptoms prior to irradiation. Three patients who died of other causes, are considered as cancer deaths. Case 22 could not be traced after 15 months and is counted as dead. Eleven patients are free of disease January 8, 1960.



STAGE	I	3/3	100%
"	II	6/9	66%
"	III	2/6	33%
"	IV	0/4	0%

Fig. 10. Study of results in 22 cases treated more than two years ago. All cases, epitheliomas, lymphosarcomas and lymphoepitheliomas are included. Solid dot represents tumor arrest; circle represents death. Note the good correlation between stage and tumor arrest. Note that a massive primary tumor without nodes (T4, NO) may have a worse prognosis than a smaller tumor with small nodes (T2, N1).

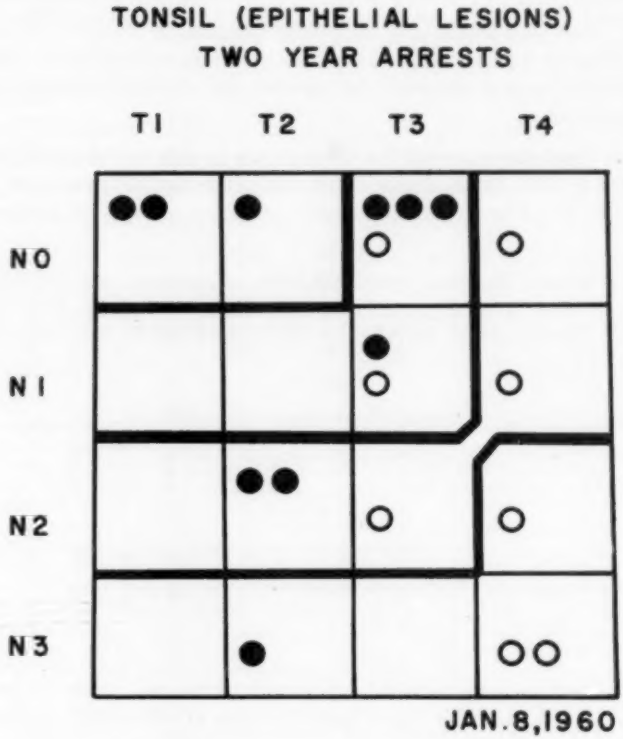


Fig. 11. Study of results among 18 epitheliomas. The solid dots represent two-year arrests; the circles, deaths. Once again is seen the good correlation between stage and tumor arrest.

ion, that a massive primary tumor without nodes (T4, NO) has a worse prognosis than a smaller primary with small nodes (T2, N1), seems to be upheld by the analysis in Fig. 10.

The 18 epitheliomas were analyzed (see Fig. 11). This analysis again showed the correlation between stage and prognosis.

A final summary of the 29 patients in this series (all histologic types), treated more than one year ago, is presented in Table II. The three year apparent arrest rate is 44 per cent

TABLE II.

Survival in All Cases of Malignant Lesions of the Tonsil.
Final summary of all cases in this series (all histologic types), at risk more than one year.

Year of Initial Treatment	New Cases	Years Arrested 1	2	3	4	5
1952	2	2	–	–	–	–
1953	1	–	–	–	–	–
1954	3	3	2	2	–	–
1955	5	5	5	4	4	1
1956	7	6	3	2	1	–
1957	4	2	1	–	–	–
1958	5	4	–	–	–	–
1959	2	2	–	–	–	–
Cases at Risk	29	27	22	18	11	6
Number Arrested		24	11	8	5	1
Per Cent Arrested		89	50	44	45	16

January 8, 1960.

and the five year rate is 16 per cent; however, these figures are not statistically significant because of the small number of cases.

From the analysis of this small series of cases treated with supervoltage rotation technique, we have not demonstrated a significant improvement in the cure rate; however, our clinical experience convinces us that supervoltage rotation technique is an improvement over conventional methods by virtue of its sparing of tissues and the lessening of discomfort to the patient.

CONCLUSIONS.

1. Cancer of the tonsil is a clinical entity, biologically dif-

ferent from cancer arising in adjacent sites, such as tonsillar pillar and retromolar fossa.

2. A method of staging cancer of the tonsil has been described, following the T.N.M. system recommended by the International Union Against Cancer.

3. Treatment of cancer of the tonsil is primarily by radiation therapy; surgery plays an important but ancillary role in the management of failures and complications of radiation therapy.

4. Supervoltage rotation irradiation has the advantage of reducing the severity of radiation reactions and provides an efficient means of delivering the necessary tumor dose to the primary tumor and metastatic neck nodes in continuity.

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CRITERIA FOR OTOSCLEROSIS SURGERY AND FURTHER EXPERIENCES WITH ROUND WINDOW SURGERY.*†‡

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INTRODUCTION.

Since the inception of Lempert's[1] fenestration procedure for correction of hearing impairment incurred by otosclerosis many changes in the corrective procedure have come about. We are no longer restricting our efforts to by-passing the pathology produced by otosclerosis. Our efforts have turned to replacing the pathological structures with artificial ossicular chains and stapedial footplates.

The limitations inherent in the fenestration procedure are no longer a problem. The new techniques evolved from Rosen's[2] original concept are not limited by the complete absence of an ossicular continuity or by the restrictions that accompany a derangement of the phase relations between the oval and round windows and the transmission characteristics of the tympanic membrane and tympanic cavity. The moderately limited success that accompanies fenestration surgery has been established by time and experience. Whereas the success that accompanies oval window surgery, whether it be stapedial mobilization or stapedial replacement is still to be determined and tested by time.

Theoretically the success of fenestration surgery was handicapped by changes in middle ear physiology which had no

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chance of elimination. Because the drum-head and ossicular chain are by-passed the average patient is left with a 20-25 db air-bone gap. On the other hand, oval window surgery offers the possibility of nearly complete, if not complete, restoration of middle ear function. On the one hand the defects created by fenestration surgery were fixed, and the surgeon was forced to accept the limited success. In the case of oval window surgery, however, new techniques and improvement in the materials used to substitute defects in the ossicular chain offer a real chance of complete correction of the middle ear deficiency created by otosclerosis.

INDICATIONS FOR SURGERY IN OTOSCLEROSIS.

When fenestration surgery was the most commonly used method of correcting the hearing defect produced by otosclerosis, certain criteria were used to determine the suitability of the cases.

It is generally recognized that the upper level of normal hearing for everyday speech is 15 db at the principle speech frequencies 500, 1000 and 2000 c/s. It is also generally accepted that when an average of these three frequencies reaches 30 db the individual will have enough difficulties hearing and understanding everyday speech to require some kind of assistance. In view of the magnitude of the fenestration procedure and the limitations placed upon the patient by the postoperative care and restricted activity, fenestration surgery was seldom done unless the anticipated result assured a threshold of at least 30 db at the speech frequencies. Practically this means that unless the surgeon can be sure of reaching a hearing level that will require no amplification to assure practical hearing for speech he will seldom perform fenestration surgery. The usual criteria for fenestration surgery were a preoperative air-bone gap of 30 db or more and bone conduction hearing levels no higher than 20 db at 500 and 1000, and 30 db at 2000 c/s.

Obviously these criteria limited the number of otosclerotic patients who were suitable for surgery which in turn severely limited the usefulness of fenestration surgery for otosclerosis.

Obviously if temporal bone surgery was to correct all the problems created by otosclerosis, methods, whose indications were less restricted and whose contraindications were not a problem, must be developed.

Rosen's technique later amplified by Shea[3] seemed to produce better hearing levels and better air-bone gap closures with few of the disadvantages of fenestration surgery; however, we believe that the usefulness of oval window surgery has been severely restricted by the use of criteria or indications originally established for fenestration surgery.

If the full benefit of this modern technique is to be realized the question of criteria must be carefully reviewed with the advantages and disadvantages of oval window surgery in mind. We believe the criteria used for selecting patients for fenestration surgery are not suited to oval window surgery and that skillful surgeons should extend their efforts beyond the limits imposed on the basis of past experience with fenestration surgery. The principles and concepts of fenestration surgery are not comparable to those of oval window surgery, and before criteria for selection of patients are established the therapeutic possibilities offered by oval window surgery must be thoroughly investigated and understood.

We draw your attention to some of our recent thinking about this problem. Several important questions are immediately brought to mind: first, should there be a maximum air conduction threshold above which surgery should not be attempted? Second, should there be a maximum bone conduction threshold above which surgery should not be attempted? Third, should a hearing aid be used to supplement the benefits of surgery?

Theoretically, regardless of the maximum air conduction and bone conduction loss, if there is an air-bone gap, reestablishing the ossicular function should produce a better hearing level, at least equal to the true sensory-neural hearing level. Measurements of high air conduction thresholds and particularly high bone conduction thresholds are restricted because of equipment limitations. When air conduction levels of 85-95 db are encountered it is frequently impossible to

obtain bone conduction responses with our present equipment. It is entirely possible that many of these cases have air-bone gaps of as much as 35-40 db. Assuming that this is true, it is quite probable that oval window surgery will close this gap and make it possible for these patients to wear a hearing aid. Results of this kind are much more important to these patients than to patients with a 30 db gain whose threshold levels are 35-45 db.

These broader concepts open up greater possibilities for the rehabilitation of impaired hearing patients than have ever been possible before.

CLINICAL PROGRESS OF OTOSCLEROSIS.

Otosclerosis can produce a pure conductive hearing loss with maximum hearing levels of 50-60 db in the speech frequencies. Otosclerosis also can produce a mixed type hearing loss whose maximum levels are really not thoroughly established. Whether otosclerosis produces a pure sensory-neural hearing loss is still a controversial subject. Those who believe this condition exists are hard put to explain its causal relations. A better understanding of the sensory-neural and mixed hearing loss found in patients who obviously have clinical otosclerosis will undoubtedly lead to new methods for correcting the impairment found in these individuals.

Diagnosing otosclerosis, when no bone conduction responses are found and there is a profound loss by air conduction, is very difficult. If the patient gives a history of a slowly progressive hearing loss the most likely diagnosis is otosclerosis. Other possibilities such as ototoxic drugs, viral disease, Meniere's syndrome, auditory nerve tumors and syphilis must be considered; but these conditions are rare compared to otosclerosis.

It has been known for several years that the effects of otosclerosis on the temporal bone can be seen by X-ray. Recently Compere[4] has emphasized this again. In our opinion, if profound hearing loss is not accompanied by increased density in the temporal bone demonstrated by X-ray the diagnosis of otosclerosis is highly improbable.

When a diagnosis of otosclerosis is made in a patient with a profound hearing loss (greater than 80 db) we believe the middle ear should be explored. These explorations have led to a number of interesting observations that need further study.

There are no important contraindications to middle ear explorations. Patients with profound hearing loss are in dire distress and help of any kind is important to them. If there is even a slight chance that surgery will improve the hearing level, middle ear surgery should be carefully considered.

SURGICAL AND HISTOPATHOLOGICAL OBSERVATIONS IN ADVANCED OTOSCLEROSIS.

The following are a few of the interesting observations we have made during the past three years:

First, some of these patients have normal appearing stapes, normal oval and round windows and when an opening is made in the footplate the perilymph appears to be normal. The hearing remains unchanged after surgery. Unless the diagnosis of otosclerosis based on X-ray findings is incorrect in these cases the loss must be due to pure sensory-neural pathology. If otosclerosis is the precipitating cause the site of the pathology must be outside the middle ear. Bruhl[5] in 1910 and Guild[6] in 1954 reported histologic evidence of otosclerosis in areas other than the middle ear in the temporal bones of patients with profound hearing loss.

Several patients have been observed with normal middle ear structures, but when pulsating pressure is applied to the stapes no round window reflex is seen. There is no substantiated explanation for this, but it appears that some sort of obstruction must be blocking the continuity of motion between the two windows.

Two patients were seen with normal oval and round windows, but the perilymph was thick and gelatinous. Siebenmann[7] reported finding cell free exudate in the cochlea of a patient with otosclerotic closure of the cochlear aqueduct in 1911.

Some patients have been found with all degrees of stapes fixation from easily mobilized ligament fixation to complete obliteration of the oval window by otosclerosis. The round window of these patients may be normal, constricted with a normal or calcified membrane, or totally obliterated along with part of the scala tympani, by otosclerosis. We have observed no patient with a normal stapes and oval window, who showed closure of the round window.

The histopathology of stapes fixation in otosclerosis is well documented in the literature starting with the first observations of Valsalva[8] in 1735. Nager and Fraser[9] published the histologic findings of six cases showing otosclerotic closure of both windows in 1938. They point out that closure of both windows is found only in patients who have total or almost total hearing loss. They point out also that there are very few of these cases. In general these cases show diffuse otosclerosis with some very active areas. It is rare, indeed, for otosclerosis to invade the labyrinthine spaces. In the six cases reported by Nager and Fraser, five showed closure of the cochlear aqueduct. All cases showed total closure of the round window.

Troltsch[10] in 1873 reported a 16-year-old profoundly deaf case with a fixed stapes and a constricted round window niche. The round window membrane was white, thickened and completely calcified.

Habermann[11] reported a case with total otosclerotic closure of the round window but no involvement of the stapes or oval window. This case demonstrated a closed connective tissue tract leading from the tympanic cavity lining to the scala tympani. The patient showed almost total loss of hearing in one ear and minimal loss in the other ear. The totally deaf ear showed complete closure of both windows. Habermann concluded from this that the round window was of little importance to hearing.

Goodhill, et al.,[12] was able to show a loss of approximately 10 db for bone conduction and air conduction in the speech frequencies when the round window in animals was closed artificially. Wever and Lawrence[13] had great difficulty

producing a hearing loss in cats by blocking the round window. They concluded that in the presence of normal middle ear structures, blocking the round window would affect the high tones more than the low tones. Massive blockage of the round window raised the threshold only 20 db in any of the frequencies tested.

We have not observed any patients with otosclerotic closure of the round window only. The effect of round window closure on the pure tone threshold and the speech discrimination score is not known.

SURGICAL TECHNIQUES.

One year ago a preliminary report[14] was published on oval and round window surgery in extensive otosclerosis. As indicated in this report the first cases were done through an endaural incision. This approach made it possible to use the dental drill to remove the thick otosclerotic bone at the windows. Subsequently, local anesthesia transcanal techniques were developed. We felt that opening both windows at the same time resulted in excessive acoustic trauma and loss of perilymph; therefore, we changed to a two-stage procedure: the oval window was opened first and then the round window a month or more later. Since the original report five two-stage procedures have been completed.

The first stage operation may be exploratory only, depending upon the findings. As indicated above, we may find no window pathology to massive involvement of both windows. If minimal fixation is found with a blue footplate, the head and crura of the stapes are removed, the footplate is then shattered and polyethylene strut is placed between the incus and footplate which may or may not be covered with gelfoam.

If massive otosclerosis is found, it is removed with a dental drill, and the open oval window is covered with either a vein graft of gelfoam, after which a polyethylene strut is placed between the lenticular process of the incus and the oval window.

The above method is a standard procedure for re-establish-

ing the ossicular chain continuity in all degrees of conductive or mixed otosclerotic hearing loss.

At the second stage of the operation the round window was opened. Local anesthesia with a transcanal approach was used. Round window closure can vary from calcification of the round window membrane to complete obliteration of the niche, the membrane, and basal part of scala tympani. In some cases the round window niche appears to be filled in, but usually a fibrous tract remains extending from the tympanic mucosa to the scala tympani. This fibrous tract can be used by the surgeon as a guide for drilling away otosclerotic bone to reopen the scala tympani. In other cases a smooth depression in the promontory is all that remains of the round window. In these cases the landmarks to the scala tympani are completely obliterated.

When opening the round window or scala tympani with a bur, it is important to stay inferior (toward the hypotympanum) to avoid the scala media and medial (toward the brain stem) to avoid the scala vestibuli, since it is on the medial side of the scala tympani throughout most of the basal portion of the cochlea. The round window was considered open when perilymph escape was noted which transmitted pulsation movements from the oval window.

The new round window opening has been closed with gelfoam, vein graft and middle ear mucosa. We do not feel this is an important factor since the endosteum probably quickly seals over the opening.

CASE REPORTS.

Case 1. (See Fig. 1.) This patient is a 42-year-old baker with a hearing loss for 15 years. X-rays show bilateral petrosal sclerosis. Both ears had approximately the same loss. In the right ear the preoperative speech reception threshold was 87 db with a discrimination score of 18 per cent. There was a definite air-bone gap with bone conduction response at 15 db for 250 c/s, 30 db at 500 c/s, 50 db for 1000 c/s and no response for bone above this.

In January, 1959, the oval window was found to be fixed with hard white otosclerotic bone. This was thinned with a bur, fractured and depressed into the vestibule. A polyethylene strut was inserted between the incus and the depressed footplate.

Following surgery the bone conduction remained the same, but the speech reception threshold improved to 62 db. Speech discrimination

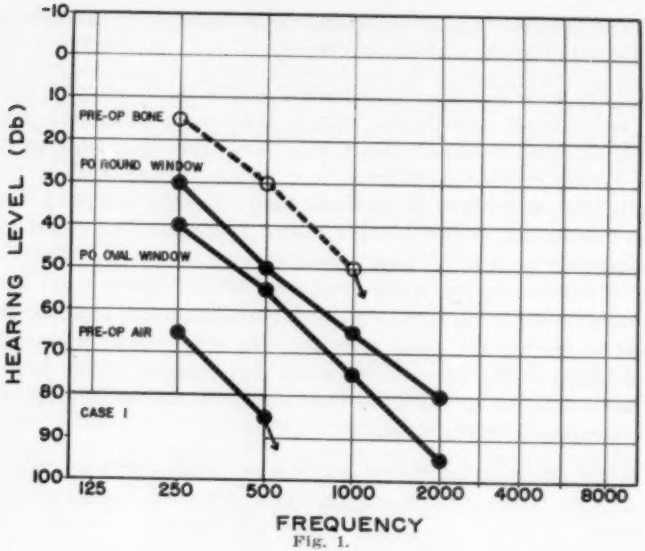
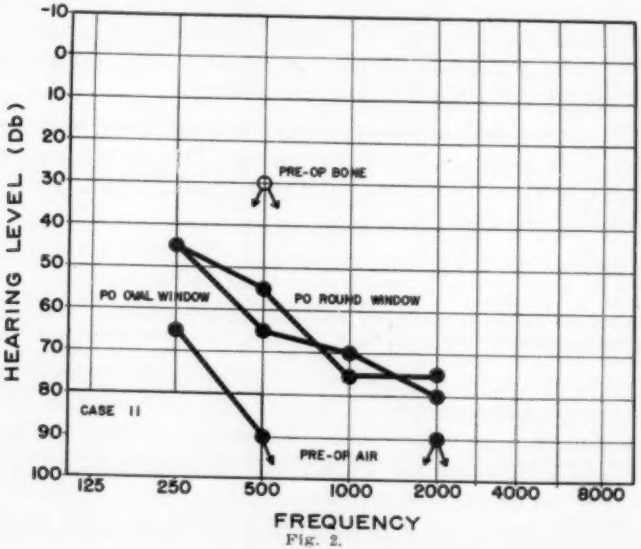

-10
0
10
20
30
40
50
60
70
80
90
100
HEARING LEVEL (Db)
PRE-OP BONE
PO ROUND WINDOW
PO OVAL WINDOW
PRE-OP AIR
CASE I
125
250
500
1000
2000
4000
8000
FREQUENCY

Fig. 1.


-10
0
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90
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HEARING LEVEL (Db)
PRE-OP BONE
PO OVAL WINDOW
PO ROUND WINDOW
CASE II
PRE-OP AIR
125
250
500
1000
2000
4000
8000
FREQUENCY

Fig. 2.

remained approximately the same at 16 per cent. One month later in February, 1959, the round window was opened. In this case it was necessary to remove only a small amount of bone. Clear perilymph escape was noted, and this moved in opposite phase to incus pulsation. There was a 20 db drop in hearing on the table.

The hearing was depressed considerably at first and has gradually improved. In December, 1959, the bone conduction was the same as preoperative and the speech reception threshold had improved to 52 db with a discrimination score of 26 per cent.

Comment: Opening the oval window was of definite benefit. In this case the round window could be opened with little dif-

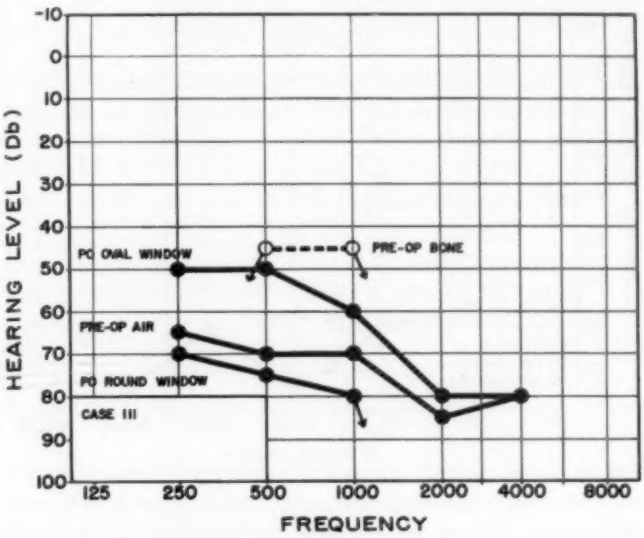

Fig. 3.

ficulty; however, it took a long time to regain the level attained by the oval window surgery alone.

Case 2. (See Fig. 2.) A 54-year-old housewife with hearing loss for 20 years had a preoperative speech reception threshold of 97 db with bone conduction of 30 db at 500 c/s only. In January, 1959, a three-quarter solid footplate was removed and a P. E. strut and vein graft placed. Postoperatively the pure tone and speech reception thresholds varied from test to test but finally stabilized at about 70 db. Six months later in June, 1959, the round window was opened. Considerable bone had to be removed. It is interesting that in the first hours after surgery the patient's hearing was subjectively improved, and her tinnitus subsided.

By the third day, however, the hearing had returned to preoperative level and has remained here since.

Case 3. (See Fig. 3.) The patient is a 32-year-old oil worker with a hearing loss of 12 years' duration. Preoperatively in the left ear, he had an S.R.T. of 68 with a discrimination score of 74 per cent. B.C. was 45 db for 500 and 1000 c/s. In January, 1959, a partially blue footplate was fragmented and a P.E. strut placed from the incus to footplate. Three weeks postoperative the S.R.T. was 48 db, but the discrimination score had dropped to 54 per cent. There was no change in B.C.

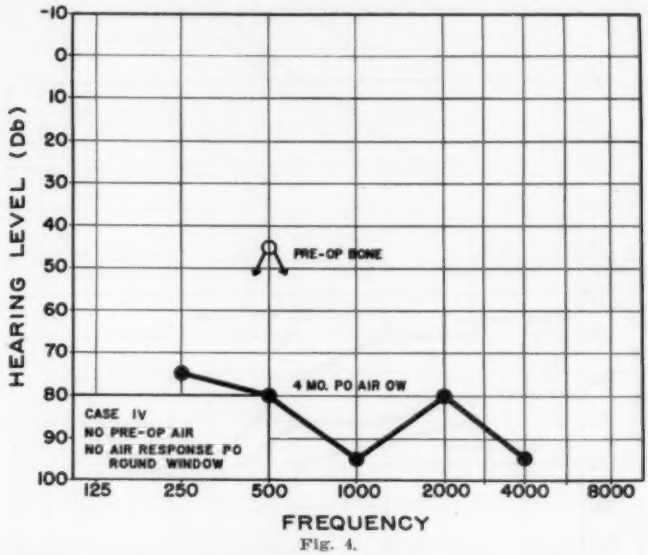

Fig. 4.

In February, 1959, the round window was opened. It was not firmly fixed but definitely had a calcified membrane. Following the procedure the B.C. has remained the same, but the threshold has dropped to 85 db where it has remained.

Case 4. (See Fig. 4.) The patient is a 57-year-old furniture finisher. He had a hearing loss of 15 years' duration. Hearing test in the right ear gave no A.C. response and B.C. at 45 db for 500 c/s only. In March, 1959, a completely solid footplate was thinned, fractured free from the oval window margin and depressed into the vestibule. This was covered with gelfoam and a P.E. strut placed. The hearing improved to a 55 db level by A.C., but the B.C. remained unchanged. The patient, however, continued to have an 0 discrimination score.

Four months later in July of 1959, the round window was opened. It was completely solid and considerable bone was removed along the basal coil before perilymph escape was noted. Pulsation pressure from the oval window was well transmitted to this opening. Postoperatively the

B.C. frequency at 500 c/s has remained the same but all response to air conduction has disappeared.

Case 5. The patient is a 52-year-old carpenter. He has had hearing loss for 40 years. He had no response by air or bone conduction in the left ear. In June, 1959, a solid footplate was removed with a drill. Periosteum taken from the ear canal was placed over the oval window and a P.E. strut placed. One month postoperative, he had a speech reception threshold of 90 db but 0 discrimination. He now also had one response by B.C. of 50 db at 500 c/s. In July, 1959, the round window was opened. Fibrous tissue tract led from the tympanic mucosa to the former round window. Perilymph with good transmitted movement from the oval window was noted. The mucosa of the promontory was placed over the opening. Postoperatively the patient has again lost all responses by A.C. and B.C.

COMMENT.

In the above five patients there was definite improvement in hearing threshold after opening the oval window. We have noted in this group and in other deaf patients on whom oval window surgery has been performed that it usually takes up to six months for the patient to transfer his aid from his formerly better ear to the operated ear.

At first there is often a very narrow loudness tolerance (usually only 10 or 15 db between threshold and pain). This gradually improves so that the patient can wear an aid with comfort. The reason for this phenomenon is unknown. Usually, if he is wearing an aid on the unoperated ear, he finds it too loud and, therefore, unbearable to wear in the operated ear.

Shortly after surgery the discrimination score is often worse than preoperative. This, too, improves with time. During the first postoperative months we usually advise these patients to have a set of earphones with an adjustable volume attached to the television. When the patient finds through practice with his ear phones that his hearing is stabilized, his tolerance increased, and his discrimination "re-learned" in the operated ear a hearing aid evaluation is done to determine the aid that is now best suited for him.

The results of these cases has led us to the conclusion that opening the oval window in conductive or mixed loss is potentially a worthwhile procedure regardless of the level of the bone conduction or air conduction. The limiting factors in

selection of cases seems to be only the width of the air-bone gap, hearing level of the other (better) ear, age, and general condition of the patient.

What about surgery on the round window? This is a potentially dangerous procedure since in three out of five of our cases the hearing was made worse. The reason for this is obscure, but probably the scala media was surgically invaded since only obscure surgical landmarks are present.

Previous case reports, animal work, and the first two cases reported in this study all indicate that the round window has little to do with hearing threshold; therefore, apparently the patient has little to gain and the chance of damage is great by surgery on this window. For these reasons we are abandoning further surgery on the round window or scala tympani until better diagnostic methods and more precise surgical techniques are available.

Several facets of otosclerosis have become increasingly apparent from clinical, radiological, and surgical study of otosclerosis.

First, the reason for a conductive hearing loss in otosclerosis is stapes fixation in the oval window. In general if the fixation is ligament in type (that is, without an actual bony bridge between the footplate and the oval window, but merely a thickening of the membrane) the amount of the air-bone gap tends to be less. If there is bony fixation, the gap tends to be 30 db or more. Once the annular ligament is bridged the audiogram does not tend to reflect the amount of the otosclerosis in the footplate or oval window.

Second, fenestration surgery, or complete release of stapes ankylosis often improves the bone conduction. This was attributed to a change in cochlear hydrodynamics by Juers.[15]

It seemed reasonable that total closure of both windows would completely immobilize the cochlear fluids and, therefore, lead to total hearing loss. By opening both of these windows it was hoped that dramatic hearing improvements could be obtained; this has not proven to be the case.

Apparently fixation of the oval window can be blamed for the amount of bone conduction loss commonly known as the Carhart notch.[16] Closure of the round window accounts for possibly only 10 db of bone conduction loss. These two figures then clearly represent the hydrodynamic changes in the cochlea caused by window closure, that can be measured by bone conduction audiometry.

The still completely unanswered question then is: Why do patients with otosclerosis develop the sensory-neural loss of mixed and perceptive otosclerosis? Bone conduction measurement is the present method of evaluating this loss. This is inadequate, because it does not separate the hearing loss due to hydrodynamic factors of otosclerosis, and it does not delineate the site of the sensory-neural lesion.

There is much to be done, we have only begun the conquest of otosclerosis.

CONCLUSIONS.

1. At present there seems to be no maximum bone conduction thresholds to contraindicate oval window surgery in otosclerosis.

2. The minimum air-bone gap that is practical to attempt to improve by oval window surgery has not yet been established. This seems to be the prinicpal limitation on the indications for oval window surgery.

3. Otosclerotic patients with stapes fixation and hearing loss beyond the limits of present audiometric measurements can be improved by oval window surgery, providing an unmeasurable air-bone gap is present. Diagnostic procedures for these patients should be improved.

4. Patients with extensive otosclerosis often have closure of the round window. Opening this window with a drill through a transcanal approach has not proved beneficial, and in three cases it has been detrimental. We are, therefore, abandoning round window surgery until more precise controlled methods can be developed.

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SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

The Sixth International Congress on Diseases of the Chest will be held at the University of Vienna from August 29 to September 1, 1960.

VERTIGINOUS EPILEPSY.*†

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That dizziness may accompany an epileptic seizure has long been known. Whether it can in itself, without other features of epilepsy, constitute a seizure, is a much more difficult problem to solve. Interest has recently been aroused in the problem by physiological stimulation of the cerebral cortex and by the occurrence of seemingly pure vertiginous seizures. It seems advisable to attempt to evaluate the state of our present knowledge concerning the problem, though it is meager and inconclusive in many important respects.

NOMENCLATURE AND CLASSIFICATION.

The seizures in question have been referred to as "epileptic vertigo," "vestibular epilepsy," "vestibular seizures," and "vestibulogenic seizures." The term vertiginous epilepsy seems preferable, since it is non-committal regarding the origin of the seizures, and it refers to their outstanding characteristics.

Vertiginous seizures may occur as an aura, preceding a seizure; or they may constitute the seizure itself. Dizziness as an aura constitutes no diagnostic problem; its interest is largely physiological, as an indicator of the possible source of the cortical discharge. When it occurs as an aura, it is followed by the typical features of an epileptic seizure which indicate its true significance. It may occur in petit mal, grand mal or psychomotor seizures. In his extensive study of auras, Gowers[1] found it to be present in 25 of 155 cases of

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epilepsy. It is one of many special sensory auras and is not infrequently encountered.

The seizures referred to as vestibulogenic seizures are in reality examples of reflex epilepsy, rare in their incidence as compared with other forms of epilepsy. They are equivalent to musicogenic or photic seizures which are initiated by auditory and visual stimuli, and they are reported to be precipitated by vestibular stimuli.

Vertiginous epilepsy, also referred to as vestibular seizures, constitutes a small percentage within the epilepsy group, and is characterized by the occurrence of vertigo or dizziness which constitutes the sole feature of the disorder. It has features of its own which I shall describe presently.

I have confined my discussion to human cases, because frankly I do not know what to do with animal experiments. The evidence for vestibulocortical projections has been conflicting to say the least. It was first considered to be ipsilateral, then contralateral and finally bilateral. It is now felt to be chiefly contralateral. It has been suggested that each vestibule projects to both reticular complexes, each reticular complex receiving afferents from both sets of vestibular nuclei.

REPORT OF A CASE.

The problem of vertiginous seizures is clearly illustrated by the following case: This was a man of 23 years, who had had dizzy spells for seven years, since he was 16 years old. They occurred at first once a month, but for two weeks before he was seen, they were an almost daily occurrence.

The episodes appeared without warning and were characterized by a sense of spinning lasting for about 10 seconds, followed by a lapse of consciousness with drooping of the head and with complete loss of contact with his surroundings. He described this in the following terms: "I forget everything for a couple of seconds, and everything is black." If he is engaged in conversation, the thread of his conversation is broken. On two or three occasions he had a dizzy spell while driving, but did not lose control of his car. He reported that he returned to consciousness suddenly with no ill-effects except for a feeling of fatigue which lasted about ten minutes. He had had no other types of seizure, and he denied previous seizures or a family incidence of convulsions or fainting spells. There was nothing of significance in his past history, and both physical and neurological examination were negative.

Laboratory examinations revealed little of significance. He had a blood count suggestive of infectious mononucleosis. The Hct. was 45

per cent and Hgb. 14.9 Gm. The leucocytes numbered 6,000, with a differential of neutrophils 30 per cent, lymphocytes 50 per cent, atypical lymphocytes 13 per cent, monocytes 47 per cent, eosinophiles 2 per cent and basophils 1 per cent. The spinal fluid examination was negative. Pneumogram revealed no evidence of tumor. The only significant findings were revealed by the EEG, which showed evidence of general paroxysmal cerebral dysrhythmia, with right temporal lobe discharges in the sleep record.

CLINICAL FEATURES OF VERTIGINOUS EPILEPSY.

That vertigo may appear frequently as an aura, to be succeeded by one of a variety of seizures, is well-known. That it may occur, however, in pure or relatively pure form, as the only feature of a seizure, is not sufficiently recognized, and for this reason it may be helpful to review its clinical characteristics.

The *incidence* of vertiginous epilepsy is probably rare when one considers the great variety of seizures and the many forms of epilepsy, both idiopathic and symptomatic. There appear to be no reliable statistics of its occurrence.

A group of 14 patients with epileptic vertigo has been reported by Pedersen and Jepsen,[2] but I have not included all of these in my discussion because in only six was there impairment of consciousness, while two had slightly impaired hearing. Eight had abnormal EEG patterns. Probably only those with impairment of consciousness should be included.

The *clinical features* of this type of epilepsy are distinctive, but not in themselves diagnostic of the condition. The central characteristic appears to be recurrent attacks of dizziness associated at time with lapses of consciousness, which, however, is not always present. In its absence there may be complete amnesia for the episode, or there may be no impairment or loss of consciousness of which the patient is aware. The lapse of consciousness covers only seconds, and is quickly over. There is no fall; objects are not dropped during the episode; and no other associated symptoms are usually present. The onset is sudden, without warning of any type. The vertigo itself varies in degree, but in quality it is always associated with a sense of movement of the patient or his surroundings. It may be typically paroxysmal in type. Nau-

sea and/or vomiting are not usually present; and cochlear symptoms are absent. That master of medical observers, Sir William Gowers has given a description of the seizures which leaves no room for improvement. He says, "The attacks of minor epilepsy which are characterized by vertigo have to be distinguished from other forms of sudden giddiness. In some cases of epilepsy, vertigo is the only symptom of which the patient is conscious, and may be the only symptom of which we can obtain any account, although vertigo is usually succeeded by transient loss of consciousness, obvious to others, but unknown to the patient himself. Other forms of vertigo, to whatever due, are rarely attended by loss of consciousness and are usually followed by long-continued giddiness, which persists while the patient is recovering. In epilepsy there is no consecutive giddiness; the patient is at once well, or merely presents mental dullness for a few minutes." It is this absence of after-discharge that constitutes the most striking distinction between a vertiginous seizure, and vertigo of some other origin. When the epileptic with vertigo has recovered from his seizure, he is immediately free of dizziness, unlike the condition found in vertigo of vestibular or central nervous system origin due to other causes.

In addition to the nature of the vertigo, impairment or loss of consciousness, or amnesia for the duration of the attack is an essential characteristic of the seizures. As in the case of a patient with petit mal, the patient with vertiginous seizures has a momentary lapse of consciousness. He may or may not be aware of this, but he may be seen to look blank or pale for the short duration of the attack. Where there are true vertiginous seizures, persistent questioning will reveal impairment of consciousness, though this may be denied by the patient.

The physical and neurological examinations in patients with the characteristics noted, are negative. This is true even if the vertiginous seizures are associated with brain disease, since the lesion responsible for the condition is so small that nothing is to be found neurologically.

The EEG may be normal, or there may be evidence of dis-

charge from one or both temporal lobes in sleep EEG. These may also be evidence of generalized paroxysmal cerebral dysrhythmia. In any event, the EEG findings are important, for without them the diagnosis becomes quite difficult.

Cortical Localization of Vertiginous Epilepsy. The localization of the lesion associated with vertiginous seizures is not specific, but what evidence is at hand in human cases indicates that the process is in the temporal or temporo-parietal region. The lesion has been found to be in the posterior half of the superior temporal gyrus, or at the parieto-temporal junction (Penfield and Kristiansen[3]). Electrical stimulation of these areas produced vertiginous experiences similar to those of spontaneous seizures (Penfield and Rasmussen[4]; Penfield and Jasper[5]; Penfield[6]). Penfield[6] reports that in seven of 108 patients who had exploration of the temporal lobe, typical labyrinthine sensations were produced on cortical stimulation. The area from which this sensation could be produced was in the posterior part of the first temporal convolution, adjacent to the transverse gyrus of Heschl. In other instances the lesion responsible for the seizures has been found in the middle rather than in the posterior part of the temporal lobe.

DIAGNOSIS OF VERTIGINOUS EPILEPSY.

The diagnosis of a vertiginous aura offers no difficulties, nor is there any trouble in recognizing a vertiginous seizure when it occurs in conjunction with other types of seizures, both petit mal and grand mal and even psychomotor; there are great difficulties, however, in establishing the diagnosis in cases in which the vertiginous seizures occur in pure culture. The problem becomes one of true vertiginous attacks, usually of brief duration, indistinguishable from vertigo due to other causes by the nature of the symptom itself. Two features provide significant diagnostic clues to the true nature of the condition. These are the absence of after-discharge, characterized by the fact that the vertigo is over with immediately, without evidence of gradually subsiding dizziness or other systemic symptoms. The second feature consists of impairment or loss of consciousness, or momentary amnesia associated with the seizure. Nothing is to be found on physical

neurological examination, but the very nature of the attacks indicates a small focal lesion for which a cause may or may not be found in the past history—evidence of head injury. The EEG will show generalized or focal activity, or both; and the diagnosis will be heavily supported by positive EEG findings. As in other forms of epilepsy, however, the EEG may be normal, but the diagnosis can be established if all other features of the condition are correct.

Vertiginous epilepsy offers difficulties in recognition, but it is a possible clinical diagnosis, and it should probably be recognized more frequently. The essential points in diagnosis consist of:

1. The presence of vertigo. This is true dizziness, and it differs in no way from dizziness due to other causes. It may even be paroxysmal and thus confused with a peripheral type of vertigo.

2. Impairment or loss of consciousness, or amnesia for the attack. Without this it is difficult to see how the vertigo can be separated from an ordinary attack of dizziness. It must be differentiated from syncope, but if this is done, and it can be determined that a seizure has been experienced, one of the most important steps in diagnosis has been established.

3. The absence of auditory symptoms. Tinnitus or other ear noises may be present, often as auras, but loss or impairment of hearing should be looked upon with caution, because of the bilateral representation of hearing and the great difficulty in determining hearing deficit of central origin.

4. EEG changes. While these are not always positive in the interseizure record of any form of seizure, the diagnosis in this and vertiginous type of seizure should not be made at the present time without support from the EEG, either in the form of focal or diffuse evidence of seizure activity.

It is difficult to be sure, but it is possible that vertiginous seizures may be more common than is at present recognized.

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POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

Colby College, Waterville, Maine.

The Department of Adult Education at Colby College is offering a short postgraduate course in otolaryngology on August 3, 4, and 5, 1960. This course is designed to review fundamental principles and present recent advances in this specialty through lectures, demonstrations and panel discussions by guest physicians of national prominence. The broad content of the course should make it equally attractive to the practicing otolaryngologist and the recent graduate of an approved residency program.

It will be conducted along the general line of the courses in Occupational Hearing Loss which have been given at the college for the past eight years. The program will be under the general direction of Frederick Thayer Hill, M.D., Medical Director of the Thayer Hospital, Waterville, Maine.

The fee for the course is $75 and includes tuition, board and room. Requests for further information should be made to William A. Macomber, Director of Adult Education, Colby College, Waterville, Maine.

ANTIMICROBIALS, TONSIL AND ADENOID BACTERIOLOGY, AND BLOOD CULTURE FINDINGS BEFORE AND AFTER TONSILLECTOMY AND ADENOIDECTMY.*

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In a recent publication[1] it was noted that chemotherapy and antibiotics apparently have not eliminated or reduced the need for tonsillectomy and adenoidectomy below the age of seven years. The author called attention to the study by Rhoads, Sibley and Billings[2] who stated (p. 880), "Our experience and that of others is that the elimination of the beta hemolytic streptococcus carrier state by antibiotic therapy often is only temporary . . . . When the pretreatment flora of the throat are restored, the micro-organisms that had been temporarily suppressed are often found to have gained resistance to the antibiotic that had been used in treatment. Hence, the hazard of prolonged antibiotic therapy not followed by extirpation of the tonsils may be serious, even if the micro-organisms that were the original objects of the anti-bacterial attack are temporarily eliminated." The conclusions of their study, "Bacteremia Following Tonsillectomy" (review of pertinent reference literature, p. 879), summarized by them, follows:

Blood cultures taken just after tonsillectomy were positive in 28.3 per cent of a group of 68 patients who received no antibiotic therapy prior to tonsillectomy. The incidence of bacteremia was reduced to 5.9 per cent in a group of 20 subjects who received penicillin in a daily dose of

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600,000 to 800,000 units intramuscularly for four to ten days prior to tonsillectomy. The incidence of bacteremia in a group of 29 patients who received 600,000 to 800,000 unit doses (half this dose for children) of procaine penicillin 12 to 18 hours and one hour prior to tonsillectomy or in a small group (seven) of patients receiving 900,000 to 1,200,000 units orally daily for five to seven days prior to operation was not reduced below that of the control group. Beta hemolytic streptococci were obtained from blood cultures four times, pneumococci once, alpha hemolytic streptococci (green forming) or Gamma anhemolytic streptococci 28 times, and a combination of Beta hemolytic streptococci and Gamma anhemolytic streptococci twice. The need for several days preoperative treatment with penicillin to prevent post-tonsillectomy bacteremia is obvious.

In the control series of 68 patients who had no antibiotic treatment immediately preceding tonsillectomy, Beta hemolytic streptococci were present in 57.4 per cent of the cultures of the excised tonsils, although these micro-organisms were found in only 28.26 per cent of throat cultures taken just before the operation. Among patients receiving penicillin in single doses only the day before and the day of tonsillectomy, 31.03 per cent had Beta hemolytic streptococci in the excised tonsils, although these micro-organisms were not present in the throat cultures taken just before tonsillectomy. Beta hemolytic streptococci were found only once in the cultures from the excised tonsils of persons receiving penicillin intramuscularly each day for four to ten days prior to tonsillectomy. Most of the Gram-positive micro-organisms except micrococci and Gaffkya tetragenus were greatly reduced in number by penicillin administered intramuscularly, but gram-negative micro-organisms, such as Klebsiella pneumoniae, Aerobacter aerogenes, and Escherichia coli, were found in increased numbers in cultures from the throats and excised tonsils of these subjects.

As a result of their report, several questions of clinical interest were stimulated: If bacteremia occurs frequently (28.3 per cent Rhoads) post-tonsillectomy without preoperative antimicrobial therapy, *a.* how can it be recognized by clinical manifestations, and *b.* should prophylactic anti-microbial therapy be administered routinely pre- and post-tonsillectomy to reduce such a high incidence of bacteremia?

The circumstances of a total physician-referred practice to a large medical center offered an excellent opportunity for the study of such questions. This report is the result of tonsil and adenoid tissue bacteriology and blood culture observations on a consecutive series of 140 patients undergoing tonsillectomy and adenoidectomy by operator A during the period October, 1958, through June, 1959.

In both the study and control groups, all of the patients came from within a radius of 150 miles of Richmond, Va. These were all considered of the medical therapy resistant type. Many of them had reached the stage where the attacks

of tonsillitis and adenoiditis were closely related, sometimes occurring with less than a week of freedom between acute attacks. Some remained in the subacute state, requiring continued administration of therapeutic doses of antimicrobials until the operation could be performed.

In the control group, cases were selected solely on the basis that the history revealed they had received no antibiotics or chemotherapy for a minimum of two weeks or longer before operation.

The majority of patients in both the study and control group gave a history of five or more attacks of clinically diagnosed tonsillitis and adenoiditis. In one patient, age six years,

TABLE I.

Age Incidence.
The patients' ages were distributed as follows:

Age in Years	Study Group	Control Group
1 to 3 years	10	2
3 to 6 years	39	11
6 to 9 years	39	19
9 to 12 years	12	8
Total	100	40

the history revealed only one known attack of recognized tonsillitis at age three years before the second attack at age five and one-half years which became associated with acute nephritis. The largest number of physician-recorded attacks in a child was 32. These occurred from age six weeks to 35 months when the operation was performed.

The slightly higher age incidence in the control group six to 12 years is possibly explained on the basis of fewer or less severe attacks in the period from one to six years.

INDICATION FOR THE OPERATION.

A review of Table II shows that primary tonsil or adenoid disease leads the list as the dominant indication for the operation. In this group are included those with systemic complications remote from the upper respiratory tract, such as

kidney disease. In most of these (73), the history of sore throat involving the tonsils preceded other manifestations of systemic disease or complications.

DURATIONS OF ADMINISTRATION OF PREOPERATIVE ANTIMICROBIALS.

The administration of the antimicrobials (see Table III) was directed or supervised by the referring physician, ac-

TABLE II.

	Study Group	Control Group
Tonsil-adenoid, recurrent acute attacks	73	25
+ History of Otitis-media, recurrent	17	6
+ History of Persistent Cough and Allergy	4	1
+ History of Repeated URT Infection	3	7
+ History of Impaired Hearing, persistent	3	1
Total	100	40

TABLE III.

Number of Patients Receiving Designated Antimicrobial Before Operation.	
Chloramphenicol	4
Sulfonamides	7
Erythromycin	23
Penicillin	23
Tetracycline	34
Tetracycline + Novobiocin	9
Total	100

cording to the indications. In each instance it was felt by the physician in charge that the patient needed therapy to control or to reduce clinical evidence of active inflammation to a minimum so as to permit the patient to tolerate the operation more safely.

Eleven (see Table IV) of those who had been maintained on an antimicrobial for longer than seven days were patients who had been hospitalized for other conditions associated with acute tonsillitis and adenoiditis and had the operation performed during the hospital stay or recovery phase.

GENERAL PLAN FOR THE STUDY.

Study Group.

In the study group of 100 patients, all received oral or intramuscular antibiotics preoperatively (see Tables III and IV). Blood was withdrawn aseptically from a suitable vein immediately before operation (patient fully anesthetized under general anesthesia); the operation was performed; the tonsils and adenoids were placed in a sterile glass container and blood was again withdrawn while the patient was still asleep.

Control Group.

In the group of 40 patients as controls, preoperative antibiotics or chemotherapy had not been given for a minimum

TABLE IV.

Preoperative Administration of Antimicrobials in Days.

Days	1	2	3	4	5	6	7	Longer
Number	0	6	21	28	13	9	5	18
Total								100

of two weeks to several months, otherwise, the technique was the same.

Method.

Bacteriological examination of the specimens was undertaken in the hospital diagnostic laboratories of the Medical College of Virginia. Tonsil and adenoid tissues were received in sterile containers, within two hours of excision. These tissues were macerated under sterile conditions, employing sand and a mortar and pestle. Homogenized tissue was moistened with sterile physiological saline. Tissue fluids and saline were then aspirated for employment as inoculum to trypticase-soy blood agar, eosin-methylene blue agar and thioglycollate broth, (B.B.L.) for anaerobic and aerobic cultivation.

Blood specimens were received pre-inoculated into the standard blood culture preparation of this laboratory. These blood cultures are six-ounce prescription bottles with a perfo-

rable rubber closure, prepared as biphasic culture bottles containing both solid and liquid media. The liquid medium is 80 to 90 ml. of brain-heart infusion broth, with added para-amino benzoic acid, penicillinase and 0.1 per cent agar, (B.B.L., modified). The solid phase is provided by a slant of trypticase-soy agar, (B.B.L.). This preparation can be relied on to support growth of fastidious aerobic and anaerobic organisms, (*e.g.*, H. influenza, Bacteroides funduliformis, N. meningitidis, Cl. welchi, etc.). Blood cultures were examined daily until growth was observed, or discarded after two weeks' time.

TABLE V.

Organisms Found in Tonsil and Adenoid Tissue Specimens.

	Study Group	Control Group
Single Organisms:		
Non-hemolytic Staph.	4	0
Alpha Strep.	3	2
Hemolytic Staph. aureus	6	0
Beta Strep.	0	5
Combinations of Multiple Organisms:		
Alpha Strep. + Neisseria	7	2
Alpha Strep. + hemolytic Staph. aureus	43	9
Alpha Strep. + hemolytic Staph. aureus + Beta Strep.	9	8
Alpha Strep. + Beta Strep.	5	0
Hemolytic Staph. aureus + Beta Strep.	14	3
Miscellaneous combinations	9	11
Total	100	40

(Organisms found in miscellaneous combinations: non-hemolytic staph., E. Coli, C. albicans, Pseudomonas, D. Pneumoniae, non-hemolytic strep., Vulgarius, Paracolon species.)

OBSERVATIONS.

Tissues, as noted in Table V, produced predominantly gram-positive populations of organisms. Outstanding in frequency in these findings were staphylococci and Beta hemolytic streptococci as probable offending pathogens. In only ten instances in the study group and four instances in the control group, as can be seen in the Table, can the tissues have been considered free of pathogenic organisms (occurrence of alpha hemolytic streptococci singly or in combination only with Neisseria). Virus studies were not included.

The results obtained on blood culture study (see Table VI) were unexpected in that no positive cultures (excepting two contaminated) were obtained in the series. This finding is at variance with other reported studies.[2] Interpretation of these results is speculative. In transient bacteremia, a single sample of blood may not establish the condition. Cultures taken in the immediate postoperative period, would be expected to reveal manipulation induced bacteremia if the organisms were circulating with any frequency. The fact that these cultures were negative leads to the conclusion that there was no bacteremia of significant magnitude occurring in these

TABLE VI.

Results of Blood Culture Studies.

	Number	Positive
Study Group:		
Received antimicrobials preoperative—		
Blood culture before operation	100	0
Blood culture after operation also	35	0
Control Group:		
Received no antimicrobial preoperative—		
Blood culture before operation	40	0
Blood culture after operation also	25	1 diphtheroid 1 mold

patients. This would suggest that the surgical removal of tonsils and adenoids, even in the presence of existing chronic infection, can be performed without exciting an active bacteremia of significant numbers of organisms. It would also suggest that the administration of antimicrobials for the control of transient bacteremia before and after tonsil and adenoid operations should be on a selective basis or with indications and not as a routine procedure.

In an effort to make our study as complete as possible, we sent a copy of our results to Dr. Paul S. Rhoads[3] whose gracious reply follows:

> In regard to your letter and interesting paper, which arrived yesterday, I can not explain the discrepancy between your results and the ones we obtained. I think our technique was proper. We have no one else's series to compare our tonsil work with; but when we did similar work with tooth roots snipped off after extraction of the teeth, our percentage of positive blood cultures was right in the middle of the range of positive blood cultures reported by other workers who had done similar studies.

Our largest number of positive blood cultures was in the ones in which we used an anaerobic method[4] described in our paper and one which you did not use. This was to put the melted agar containing the patient's blood into a sterile Petrie dish, then to let it cool, after which a layer of plain agar was put on top. After this cooled, a layer of melted paraffin was put over the agar containing blood. Then when colonies appeared in the agar, it was possible to take off a small piece of paraffin and go in and get the colony with a small sterile glass pipette and bring it out for identification.

As to the fairly large numbers of Beta hemolytic streptococci found in your "study group," my guess is that these were not eliminated because the patients did not have the same routine which we used in our study. For instance, you say 23 patients received Pencillin but do not say whether it was given intramuscularly or orally and for how many days. We soon learned that one could not count on the administration of any antibiotic for several days unless he administered it himself. In the patients who received Penicillin intramuscularly preoperatively, in practically every instance we injected the material ourselves; so we know those patients received the antibiotic. As our paper indicated, antibiotics given orally and for only short intervals did not uniformly have the effect of eliminating Beta hemolytic streptococci. You w"l also note that we used a quantitative method in our cultures of the tonsils, and therefore shifts in the bacterial population of the tonsils which we described were much more meaningful (see the Table in both of our reprints).

Also, I note in your figures on page 9 (Table VI) that you had a relatively small series of patients who had blood cultures postoperatively. It is possible that had your series been as large as the series of blood cultures before operation, you would have picked up more positive blood cultures. In our report we were not trying to make the point that every patient who is to have a tonsillectomy should necessarily have five days of Penicillin administration by injection. Transient bacteremia seldom is a serious threat. Present views are that unless there are previously damaged heart valves, subacute bacterial endocarditis is not apt to be set up when the bacteremia is of very transient duration and the valves are normal. However, I do think the precaution we suggested is important in patients who have had previous valvular damage. We tried to point out in subsequent papers that there might be an even higher incidence of potentially dangerous Staphylococci in infected tonsils after the prolonged administration of antibiotics to which these organisms were resistant; and we did point out that other organisms, such as E. coli, Diplococcus pneumoniae, and so forth, were very apt to be on the increase in such persons. Thus, I do not think too much therapeutic implication should be read into our results. We were giving them for what they were worth without making too many recommendations as to what doctors should do about it. I do believe that our blood cultures are correct, however.

SUMMARY.

The administration of various antimicrobials to patients with recurrent or persistent attacks of tonsillitis and adenoiditis does not eliminate the pathogenic organism in the tonsils or adenoids in most instances (90 per cent). The fact that our study did not show any demonstrable evidence of bac-

teremia in blood cultures should in no way minimize the importance of such a possibility in the event of clinical symptoms of bacteremia before or after tonsillo-adenoidectomy.

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL.

The Gill Memorial Eye, Ear and Throat Hospital has just completed its Thirty-Third Annual Spring Congress in Ophthalmology and Otolaryngology and allied specialties. The attendance was one of the largest in the history of the school with an attendance of 300 doctors and their wives. There were 40 states, including Canada, represented. There was a total of 18 guest speakers and there were 50 lectures during the five-and-a-half days of the Spring Congress. In 1961, the Thirty-Fourth Annual Spring Congress will be held from April 9 through April 14.

MANAGEMENT OF MAXILLO-FACIAL FRACTURES.*

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The diagnosis and treatment of facial fractures form an integral part of the practice of many otolaryngologists. The literature reveals varied methods of management. Some are simple; some are complex; but each endeavors to restore the contour and immobilize the fragments. Most yield good results as exhibited by their respective proponents.

A review of the anatomy of this region is important to reacquaint us with some of the problems that are encountered and to understand better the principles and methods used to correct them.

The skull is divided into the upper or cranial region, the middle or zygomatico, maxillary and nasal region, and lower or mandibular region.

This paper will concern itself chiefly with the fractures of middle third and more especially the lateral portion. (Nasal fractures will not be considered here.) To gain a better perspective of the problem, a brief survey of the kinds and peculiarities of the fractures of the entire middle third will be discussed. This will provide a better foundation for the further discussion of the fractures of zygomatic and maxillary bones.

The orbital rim is formed above by the frontal bone, and laterally by the zygoma. The zygoma and the maxilla form the inferior margin; the maxilla then curves with the frontal to form the medial portion. At the inferior rim, both the zygoma and the maxilla continue inward as a thin plate of bone to help form the floor of the orbit or the roof of the maxillary sinus. Viewed from below, the zygoma has an

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outward projection which, with the strong abutments of the arch, produces the most prominent feature of the cheek, the cheek bone. This arch is formed by the buttress of temporal bone posteriorly and of the maxilla anteriorly. Beneath this arch lies the converging fibers of the temporalis muscle. The prominence of the zygoma blends and recedes into the anterior wall of the maxilla giving the face its bone for the cheek.

CLASSIFICATION.

A. Fractures, Lateral Region. A fracture involving the zygomatic bone and zygomatic arch without interference of mandibular movement, or a fracture of zygomatic bone with minimal displacement is classified as a first degree fracture. When, in addition to the above, the lateral wall of the maxillary antrum is involved with alteration of facial contour and interference with mandibular movement, this becomes, then, a second degree fracture. When, in addition to the above second degree, there occurs a comminution of orbital floor and/or gross separation of zygomatic-frontal suture and depression of orbital floor, this is considered a third degree fracture.

B. Fractures, Central Region.

1. The simple alveolar fracture; it is usually bilateral. There is avulsion of part of the bone.

2. The low level subzygomatic fracture (Lefort I or Guerin).

Lefort I. The low level subzygomatic involves the lower anterior wall or walls of the maxilla just below the zygoma. The fracture line commences at point on the lateral margin of the anterior nasal aperture at the pyriform sinus; it passes laterally above the canine fossa and then inclines upward to the pterygo maxillary fissure to fracture the pterygoid lamina; likewise, it also passes from the anterior nasal aperture along the lateral wall of nose to join the fracture line behind. This fracture may be unilateral or bilateral.

3. Pyramindal subzygomatic fracture (Lefort II).

Lefort II has greater involvement than Lefort I. The fracture line passes from upper end of the nasal bones at the frontal suture line down to across the frontal process of maxilla, down the lacrimal bone and laterally and downward over the inferior orbital margin at region of the zygoma-maxillary suture and then laterally near the zygomatic buttress and upward to beyond the inferior orbital fissure. This is higher than in Lefort I.

C. Combined Central and Lateral Fractures.

a. High level supra-zygomatic fracture (Lefort III).

Lefort III. This is supra-zygomatic fracture. It begins at a line above the nasal frontal suture and passes to the frontal processes of superior maxilla; it passes through upper limit of the lacrimal bone and then crosses the orbital plate of the ethmoid to down below the optic foramen and laterally to reach the inferior orbital fissure. From here it passes from the lateral wall of orbit to reach the junction of zygomatic bone with greater wing of the sphenoid.

b. The above with addition of a midline split separating maxilla into two.

c. a and b with fracture of roof of orbit or frontal bone.

From both a practical and clinical point of view it is obviously impossible to classify in every instance a fracture of the middle third of the skull in a certain specific category; however, a familiarity with each group does help to give a working knowledge of the pattern a fracture may assume.

Applied Surgical Anatomy. The immediate under side (middle third) of the cranium (upper third) is the architectual problem that is necessary to be understood.

The maxilla, zygoma, nasal cavity, orbits, sphenoid bone, and palatine bones are the integral parts of the middle third of the skull.

Maxilla. By the arching of the palate and the abutments of the maxilla against the frontal, the zygomatic and ethmoidal sutures, any given force is greatly resisted and better dis-

tributed. A further support is given by the sphenoid bone and its greater wing. The zygomatic bone redistributes the load mostly to the frontal bone and some to the temporal bone. The orbits and nasal cavity are contained within the framework in the middle part of the skull. They diminish the area available for basal bone structure. The sphenoid bone is the keystone of the skull and is situated between the facial skeleton and the cranial base.

GENERAL CONSIDERATION OF NATURE OF INJURY.

The nature of the structure of the facial skeleton permits it to resist much force from below. A blow to the anterior, superior, or lateral aspects will tend to shear off the whole complex from the cranial base.

With any maxillary bone injury there are three important associated injuries that should be considered and evaluated.

1. A gross disruption of the maxilla will displace the elements of the nasal septum and nasal bones to expose the paranasal sinuses to infection. The nasal respiratory tube may be obstructed by blood clot or displaced cartilage and bone.

2. A gross disruption of the maxilla with severe comminution will not only affect the inner canthal ligaments of the eye, but will also involve the orbital floor which in turn affects the support of the eye. Diplopia will be present immediately; and, possibly later, entropion.

3. A gross disruption of the ethmoidal labyrinth will involve the cribiform plate and give direct access to the anterior fossa of the skull.

With any zygomatic bone injury there are important associated injuries that should likewise be considered and evaluated.

With slight disruption of the zygoma the protective function of the orbital rim is impaired, and the natural prominence of the cheek is reduced.

With gross disruption of the zygoma with severe downward

displacement, there is much greater loss of the rim of orbit and the cheek bone is now flattened. In addition, the infra-orbital nerve may be crushed or torn with resultant anesthesia. If still greater disruption of the zygoma occurs, the external canthal ligament is displaced; the check ligaments are loosened; the extraocular muscles do not function properly, and the ocular level is lowered.

Acute fractures of the facial bones are not, as a rule, surgical emergencies; however, if asphyxia, shock, or hemorrhage are present, active and immediate intervention becomes necessary before any attempt at reduction and immobilization of the fracture can be done. If these three exceptions do not occur, definitive treatment should be instituted as soon as possible when the general condition permits. On the other hand, over enthusiastic and too early surgical intervention may add to the problem and may even cause death. Good surgical judgment, then, is imperative.

Management may consist of simple reduction with no immobilization and trusting that normal alignment and contour will be preserved. This may suffice if no further force or violence is encountered by the patient; however, many fractures are so comminuted and depressed that some support either external or internal or both must be applied if immobilization is to be maintained.

When comminution and depression do exist the normal cavity of the maxillary sinus is altered; *i.e.*, decreased in size, and sequestered bone may protrude into it. It is always desirable when the maxillary sinus is severely injured to use a Caldwell-Luc approach to inspect the sinus cavity for loose bone fragments. Where possible, those fragments should be replaced in their normal relationship and position. If the infraorbital rim is so shattered or disrupted, not only will it become necessary to use external skeletal fixation, but also an intra-antral packing may be essential to restore and maintain it.

One or two wires passing underneath the zygomatic arch may be required to hold out the fractured pieces. The zygoma may be so fractured that simple elevation is insufficient and

only by external support will its position be maintained. External fixation or traction to supports from a head cap may be necessary.

A careful clinical examination, as well as X-ray examination then is most important before any decision should be made as to the best method to employ. As a general rule, the fracture is more extensive than the X-ray picture usually indicates.

As stated above, there are numerous methods which the surgeon may employ to reduce and immobilize the fracture. I believe that the best method is that one which applies to that patient and his especial variety of fractures.

The following methods from the simple to the more complex are enumerated:

1. Simple elevation by hook or towel clamp, especially for maxilla.

2. Elevation by incision and fulcrum action elevator, especially for zygoma.

3. Open reduction and forceful elevation, either for the maxilla or zygoma or both.

4. Open reduction and elevation by one of the first three methods and transosseous wiring.

5. Open reduction and elevation by one of the first three methods and circumferential internal skeletal fixation.

6. Open reduction and elevation by one of the first three methods and fixation by internal wire pin stabilization and, possibly in addition, by external skeletal fixation.

7. Open reduction and elevation by one of the first three methods and intra-antral packing or balloon, or by mechanical device.

The more severely comminuted and extensive the fracture, the greater the necessity for fixation and stabilization. One patient may require two or a combination of the procedures to accomplish a satisfactory result.

It is quite evident that one may not designate a certain method as superior and applicable to all fractures. Personally, I would not like to be limited to any one procedure. It is true certain surgeons will achieve much success by using one method in preference to others where another will employ a second form of treatment with equal success.

Several examples have been selected of case histories and X-rays of the patients who have had varying degrees and types of fractures. You will probably note I have none of internal wire pin fixation. I have used that procedure earlier, but I have used other methods to a greater extent and success.

Case 1. This patient received a sharp blow with a small hammer directly to his infraorbital ridge. X-ray revealed a slight fracture of the infraorbital ridge with depression which was evident on palpation. Traction by using a towel clamp was employed to elevate the bones. A hook could have been used. No operative incision was necessary. In a fracture of this type, no further care is needed if no later force is inflicted. Healing in good position resulted.

Case 2. This patient gave the history of being accidentally hit with pipe while at work. The clinical and X-ray findings were similar to Case 1, as was the management.

Case 3. This patient received a blow by a fist; X-rays revealed a fracture of maxilla and zygoma with internal distortion and depression. There was too much ecchymoses with contusions to depend only on towel clamp or hook for elevation. By a Caldwell-Luc approach there was found to be minimal comminution present. An elevator was introduced into the sinus cavity and with manual manipulation, the fragments were replaced into normal position. No further fixation was used and the fragments remained in normal position and healed satisfactorily.

Case 4. The patient received a blow to the face by fist, probably with brass knuckles. There were marked abrasions and contusions of the face and edema of conjunctiva with subconjunctival hemorrhage. X-rays revealed a markedly displaced and fractured maxilla and zygoma. By using a Caldwell-Luc approach several small pieces of loose bone fragment within the antral cavity were removed. The maxilla revealed further extensive comminution of the remainder of the bone. An elevator was inserted into antrum and the bones elevated into position. Because of this comminution of the anterior wall and floor of orbit, a rubber glove finger filled with half-inch gauze impregnated with Amertan and covered with Amertan, was inserted into the antrum cavity after an antrum window had been made. Further packing through the antrum window opening was then done to make the intra-antral packing more firm and tight. The Caldwell-Luc incision was closed. X-ray revealed the fragments in good alignment. Healing in good position resulted. The intra-antral packing was removed in two weeks.

Case 5. A six-year-old child was struck on the cheek by an auto bumper. Her findings and management were essentially that as of Case 4.

Case 6. The patient was in an auto accident and received severe contusions of the face on the left side. Further examination revealed diplopia, marked flattening and depression of cheek bone, crepitation

and crunching on slight pressure. X-rays revealed severely comminuted fracture of zygomatico-maxillary bones. With a Caldwell-Luc exposure of antrum there was complete comminution and depression of the entire infraorbital ridge. The zygomatic prominence was easily movable. After some free and unidentifiable bone pieces were removed from sinus cavity, an elevator was inserted and lateral wall pushed outward and upward. Simultaneously, a tenaculum was fastened to the zygoma and it was gently pulled upward and laterally. After an antrum window was made, wires were placed around infraorbital ridge and under zygomatic arch. Later these were attached to support from a plaster-of-Paris head-cap. The Amertan-covered rubber finger filled with half-inch gauze was inserted and further firmly packed into the antrum cavity with its open end extending out through a previously made antrum window. This was left in three weeks and wires on the headcap for five weeks. Final result was satisfactory.

Case 7. A patient received a severe head injury in an auto accident. There was marked contusion and edema with ecchymoses. Diplopia was present. Palpation revealed emphysema and a little pressure gave a crunching sensation. By a Caldwell-Luc approach the antrum cavity was found to be almost completely obliterated and what remained was filled with blood clot and pieces of bone. The larger fragments were pushed outward as much as possible and those fragments that could be replaced were positioned. Wires were looped around the zygoma and infraorbital ridge. The antrum cavity was packed with rubber glove finger containing gauze with half-inch packing impregnated with Amertan, the open end protruding through an antrum window opening. External traction was instituted by attaching with rubber bands the wires to the metal supports from a plaster-of-Paris head cap. Gradually, over a period of three days, the zygoma and maxillary bones were drawn into position as the swelling subsided. The intra-antral packing was removed in three weeks, and the wires were removed in six weeks.

Case 8. Patient was in a fight and was hit and kicked in the face. The right side was severely contused and abraided; diplopia was present. Palpation revealed crunching of the bones on moderate pressure; neither a cheek bone nor an infraorbital rim could be felt in normal position. A Caldwell-Luc exposure revealed an antrum filled with fragments of bone and blood clot. By a tenaculum attached to the zygoma on the outside and elevator placed within the antral cavity the bones were gently pushed outward and upward into position. Some bone fragments could be replaced and others had to be removed. Wires were placed around inferior orbital ridge and around zygomatic arch. An intrasinal rubber finger with packing was inserted; the wires were fastened to suspension rods from a plaster head cap. At the end of three weeks the intra-antral packing was removed through antrum window and wires removed in five weeks. Result was satisfactory.

Case 9. Patient was kicked in the face by a horse and suffered multiple fractures of zygomatic and maxillary bones. There was marked contusion, abrasions, ecchymoses, crepitation and crunching, especially of anterior portion of cheek bone and all of anterior wall of cheek. By a Caldwell-Luc approach it was found that all of the anterior wall of maxilla and zygoma were shattered and comminuted. Before an intrasinal rubber finger filled with half-inch packing was placed within the antrum, circumferential wires were placed from the infraorbital ridge and anterior wall of the antrum to the zygoma and lateral process of the frontal bone. These were left in place for four weeks. The final result was satisfactory.

Case 10. This patient fell from a scaffold and suffered fractures of

the nose and both maxilla as well as an inwardly displaced alveolar arch. Marked contusions, abrasions, ecchymoses and subconjunctival hemorrhages were present. The nose was flattened. A Jelenko dental arch was fastened to the upper teeth; the alveolar process with metal arch was drawn forward. Circumferential wiring was suspended from zygoma to each end of metal arch and tightened just enough to immobilize bone fragments and the alveolar process.

The nose was not corrected at this time due to marked swelling of all intranasal and paranasal tissues.

The wires were left attached for six weeks, when they as well as the metal arch were removed. Healing in good position occurred.

SUMMARY.

A review of the injuries occurring in the outer portion of the middle third of the skull revealed that a variety of operative methods were used. The correction and immobilization of the fractures of the zygomatico-maxillary complex depended upon the extent of the trauma.

A practical and working knowledge of all the types of facial fractures of the middle third of the skull, coupled with familiarity with the various corrective procedures, is pertinent to insure good results.

After a careful appraisal of the character and extent of the injury, the surgeon may select that method or combination of procedures which in his hands proves most efficient.

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CORDABRASION TREATMENT OF BENIGN VOCAL CORD LESIONS.*

A Preliminary Report.

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INTRODUCTION.

Benign lesions of the vocal folds are not uncommon[1] and the technique for the removal of these tumors is well established.[2] There is, however, considerable evidence that the results have been something less than satisfactory in many instances due to recurrent or residual voice abnormality; consequently, a technique is needed which will provide: *1.* a biopsy, *2.* complete excision, and *3.* a smooth vocal fold postoperatively which would produce a pleasing voice. Unfortunately, with the present-day method of stripping the vocal cord, or excising small cordal lesions, the vocal fold is often distorted, so that the end result is chronic hoarseness or dysphonia. It was with this in mind that the "Cordabrasion" technique was developed.

MATERIALS AND METHODS.

With the experience gained in the dermabrasion procedures, an attempt was made on 16 patients with benign vocal cord lesions to perform a biopsy with the cup bite forceps followed by a cordabrasion using the technique described hereafter. The lesions were variously diagnosed as polyps, cysts, papillomas, laryngeal nodules, dyskeratoses and hyperkeratoses, chronic laryngitis, hemangiomas, and fibromas. All of the patients experienced a recovery of normal phonation, and the vocal cord regained an entirely normal appearance.

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TECHNIQUE.

Anesthesia is especially important in cordabrasion. It must produce relaxation of the vocal cords and provide sufficient time for the procedure to be completed. The patient is first given Pentothal Sodium® intravenously and hyperventilated with oxygen. This oxygenation allows a safe apneic period of three to five minutes, during which time the surgery is performed. While the mask is being removed and the laryngoscope introduced, Anectine® is administered intravenously

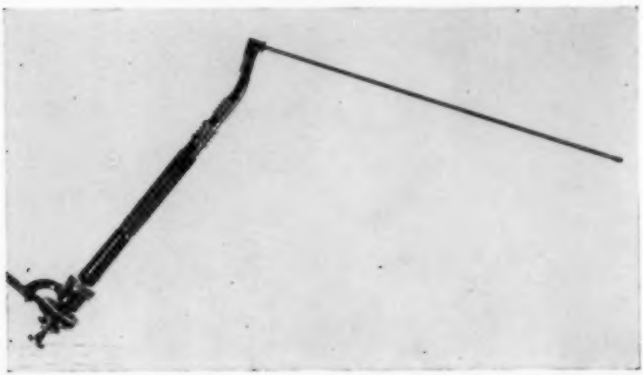

Fig. 1. Specially-designed diamond burr attached to contra-angle handpiece.

in a single injection to achieve temporary paralysis of the larynx. The laryngoscope is passed down into the laryngeal vestibule so as to be wedged at its distal end between the false cords and almost directly on top of the true cords. Biopsy-excision of bulky lesions is then done with the Cup-Bite Angular Jaw Forceps.

Thereafter, a specially-designed diamond burr* (see Fig. 1), attached to the usual drill and handpiece used in temporal bone surgery, but with a contra-angle attachment to aid visualization through the endoscopic instrument (see Fig. 2),

*Manufactured by the O'Rourke Diamond Company (Ordco), 11423 Vanowen, North Hollywood, Calif.

is introduced into the glottis. The abnormal portions of the vocal cords are abraded with this burr where indicated on the approximating and superior surfaces, smoothing out the biopsy defect at the same time. Great care is taken to avoid the vocal processes and the anterior commissure, but both cords may be "sanded" at the same surgical procedure. It was found to be more convenient to start posteriorly and produce the abrasion working forward or anteriorly.

In two patients, where the lesion was an extensive polyposis

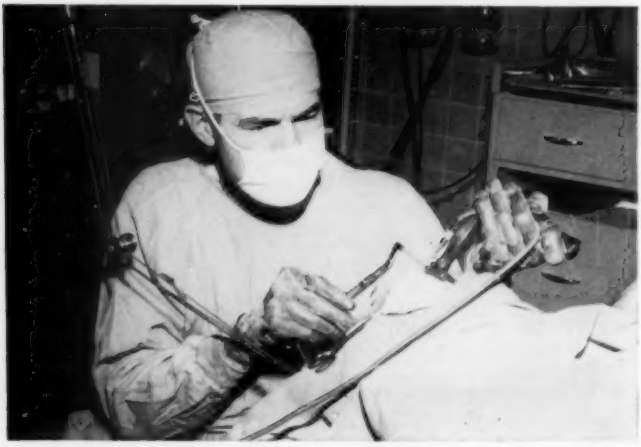

Fig. 2. Cordabrasion through anterior-commissure laryngoscope under general anesthesia.

of both cords requiring greater operating time, a small endotracheal tube was passed and left in the posterior commissure during the procedure. This, surprisingly enough, did not interfere with the exposure of the cords and, furthermore, had the added advantage of holding the cords taut in order to facilitate the cordabrasion.

Postoperatively, the patients were advised to not use their voices for two weeks, followed by a "whisper" period of another two weeks. At the end of this month, phonation was

only gradually permitted. Harmful habits, such as smoking and vocal abuse, were discouraged from the beginning.

RESULTS.

The encouraging aspect of this preliminary report is that every one of the 16 patients regained a natural, clear voice, and the vocal folds uniformly returned to a normal appearance. The technique was especially satisfactory in those cases of polyposis and dyskeratosis calling for a "stripping."

In my hands, stripping of the cords with laryngeal forceps has never been a satisfactory procedure. The true folds are too often left ragged and irregular, whereas in this method, they remained smoothly denuded.

The cordabrasion technique was found to be especially effective with early laryngeal nodules in professional singers and entertainers. The smallest of incipient vocal cord nodule or fibroma may be trimmed without taking too much of the approximating edges. A quick improvement in voice is thus obtained, and early lesions can be safely operated upon with this technique.

The wisdom of denuding both cords at the same operation has been questioned, yet by avoiding the anterior commissure, no webbing, synechiae, or stenosis resulted. By allowing only a regime of strict voice rest postoperatively there were no troublesome granulations or granulomas.[3] This being, in essence, a scarification technique, the complications of granulomata or anterior commissure webbing are considerably less likely.

COMMENT.

The primary difficulty of removing benign vocal cord tumors is that excision of a sufficient portion of the vocal fold to remove the mass may result in a dysphonia which is irreversible. Cordabrasion appears to be a possible solution.

Lowenthal[4] emphasizes the principle that removal of the lesion without damage to the underlying elastic membrane will produce a normal cord; however, herein lies the difficulty.

I have recently seen a well-known singer whose voice was permanently damaged by the removal of a papilloma from the cord in such a fashion that a large niche was left permanently in the approximating edge.

The Jacksons[5] recognized this when they warned against removing too much tissue and leaving a concavity. They believed it better to "leave a convex edge of the lesion on the cord, even if this should allow a little of the basal tissue to remain. If necessary, a secondary procedure can be done." This quotation would seem to underscore the difficulty of conventional techniques.

Again, let me emphasize that this is a preliminary report and a more accurate evaluation of the method can be obtained only after further experience.

CONCLUSION.

1. The cordabrasion technique represents a possible solution to the oftentimes unsatisfactory results in voice quality which may be obtained by laryngoscopy and biopsy-excision.

2. The technique herein described is effectively employed in the removal of the smallest of cordal lesions to the excision of extensive polyposis of the vocal cord.

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VIRAL INFECTIONS OF THE RESPIRATORY TRACT.*†

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The role of viral infections of the respiratory tract as a prime cause of morbidity throughout the world is recognized, and the impact of these diseases on civilian and military populations is rightfully regarded as an economic and medical threat of major importance. Their frequent association with bacterial complications augments their dangers. In clinical severity, they range through a broad spectrum from minor illnesses to swiftly fatal diseases, and epidemiologically they run the gamut between isolated sporadic cases and worldwide pandemics.

During the past 25 years, a great deal of information has become available concerning viruses found in association with diseases of the human respiratory tract, although many of the problems concerned with these diseases remain unsolved.[1] Unfortunately, the clinical significance of some of the more recently described viruses is not yet fully appreciated, so that classification on the basis of causative agents is inadequate. In addition, the likelihood that numerous, as yet undiscovered, viruses exist further complicates the problem. In view of these difficulties, the Expert Committee on Respiratory Virus Diseases of the World Health Organization has recently recommended a simple classification of respiratory syndromes caused by viruses[2] (see Table I). It is my intent to limit my discussion today to influenza viruses and adenoviruses and the diseases they cause, recognizing the fact that the common cold remains the most important unsolved problem in this field.

INFLUENZA.

Three antigenic types (A, B and C) of human influenza

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virus are recognized. Types A and B have undergone several changes antigenically since they were first described, although the alterations in influenza B viruses have been much less striking than those observed in influenza A viruses. No evident changes in antigenic constitution have been found in influenza C viruses. Since the first recovery of influenza A virus in 1933, minor changes have occurred from year to year, with major alterations occurring infrequently and marked by the appearance of antigenically quite different strains of virus that remained predominant. In 1946-47, A 1 influenza virus

TABLE I.

Respiratory Syndromes Caused by Viruses.

1. Influenza	Types A, B, C
2. Common cold	Unknown
3. Virus pneumonia	Psittacosis
	Adenoviruses
	Atypical pneumonia (Eaton, et al.)
4. Unclassified acute respiratory disease—	
Acute respiratory disease	Adenoviruses (ECHO; JH; 2060)
Nonbacterial exudative pharyng	Adenoviruses
Pharyngoconjunctival fever	Adenoviruses
Keratoconjunctivitis	Adenoviruses
Obst. laryngotracheobronch	CA virus (Inf.; HA 2)
Acute pneumonitis (infants)	Sendai (Adenoviruses; HA 1)
Herpangina	Coxsackie A
Pleurodynia	Coxsackie B

These data were assembled from the First Report, Expert Committee on Respiratory Virus Diseases. World Health Organization Technical Report Series No. 170, 1959.

was recognized and represented the first important antigenic change of this kind. The second and most striking change in antigenic character was observed in 1957 when the A 2, or Asian influenza virus, appeared.

The pandemic caused by the Asian virus has been described repeatedly. Following its appearance early in 1957 in China, the virus spread throughout the world during the next six months. Four phases could be discerned in its progress. The first phase was the introduction of the virus, spread by human contact, and apparently more frequently by means of shipping and rail traffic than by air travel.

The second phase was that of virus seeding. Although in some areas infection spread rapidly after introduction of virus, particularly in Europe and the United States, large outbreaks did not occur until six weeks or more following the first detection of virus.

The third phase was that of the main epidemic. Throughout the world, the morbidity was high, the mortality low, and outbreaks were often explosive. Morbidity was greatest among young children, but mortality increased with age.

The fourth, postepidemic, phase followed the main epidemic phase and was characterized by an apparent relative quiescence of infection, in an environment in which Asian virus was isolated for many months from sporadic cases.

During the pandemic of 1957, death occurred in some patients with influenzal pneumonia without any evidence of coincident bacterial infection. The histologic changes in the lungs of such patients closely resembled those seen in patients dying of influenza during the pandemic of 1918. When pneumonia occurred during the course of influenza in 1957, it was usually in patients with pre-existing chronic pulmonary congestion associated with disease of the heart and lungs or with late pregnancy. In patients dying of Asian influenza, secondary bacterial infections were common and pneumococcal and staphylococcal pneumonias were most frequently encountered, the latter being more prominent.

With the exception of atypical pneumonia associated with psittacosis, there is no specific treatment for any of the diseases of the respiratory tract due to viruses, including influenza. When influenzal pneumonia is complicated by secondary bacterial infection, appropriate antibiotic therapy is indicated; however, the indiscriminate use of antibiotics in uncomplicated influenza in previously healthy persons is unwarranted. There may be some justification for the use of antibiotics in patients with chronic pulmonary or cardiac disease who contract influenza, although this should be a matter of individual appraisal and not a routine procedure. A similar attitude of mind should condition the approach to the care of patients with acute staphylococcal infections, diabetes, neuro-

logical disorders with respiratory involvement, and in late pregnancy patients who acquire uncomplicated influenza.

The administration of 1.0 ml. of inactivated virus vaccine containing representative strains of influenza A and B viruses is the most effective method for the prevention of influenza. Immunization of children should be modified by reducing the amount of vaccine according to age and weight, and in all groups the injection of vaccine into egg-sensitive individuals must be avoided.

ADENOVIRUS INFECTIONS.

Among the recently described adenoviruses, there are at least 18 distinct antigenic types. Some of these are important causes of acute infections in man, involving the respiratory tract from the nose to the lungs, the conjunctivae, and the intestinal tract. The clinical pattern of disease depends on the extent and location of involvement. Cellular necrosis, with inflammation and hypertrophy of the regional lymphoid tissue, occurs.

The respiratory disease produced in adults by adenoviruses is characterized by headaches, malaise, and aching muscles. Fever, pharyngitis, and cough are present, and they may be accompanied by varying degrees of evidence of involvement of other tissues, including conjunctivitis, coryza, exudate on the hypertrophic lymphoid tissue of the throat, lymphadenopathy, tracheobronchitis, bronchiolitis, and atypical pneumonia. The syndromes of undifferentiated acute respiratory disease, nonbacterial exudative pharyngitis, atypical pneumonia, and pharyngoconjunctival fever fall in the group of conditions caused by these viruses. In addition to respiratory illnesses, follicular conjunctivitis, epidemic keratoconjunctivitis, and, in children, gastrointestinal symptoms may be caused by certain of these viruses.

Adenoviruses are widespread throughout the world. They may be recovered from the nasopharynx and from feces, and may be transmitted by the respiratory or gastrointestinal route. Outbreaks of acute respiratory illness caused by these agents are facilitated by crowding and poor hygiene, and

favorite sites for epidemics are military camps, particularly among new recruits, and institutions for children. These infections are especially prevalent during the Winter, and the morbidity in the Summer is usually low. At present, they are far more common among the military, although Summer epidemics of Types 3 and 7 infections have been observed among civilians, especially children.

Inactivated vaccines containing Types 4 and 7 have been proven to be very effective in preventing acute respiratory disease caused by these agents; however, the low incidence of disease in the civilian population and in permanent military personnel does not justify the use of the vaccine except among newly recruited personnel in the Armed Forces where the morbidity is high.

SUMMARY.

Infections of the respiratory tract constitute a problem of prime importance throughout the world. In spite of the tremendous advances being made in discovering new respiratory viruses, most of these infections are still undefined as far as causative agents are concerned. Much has been learned about influenza viruses and, more recently, about adenoviruses and the diseases they cause; however, the common cold still confronts investigators in this field as the most important unsolved problem, since the causative agent or agents are yet unknown.

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ENZYME THERAPY IN OTOLARYNGOLOGY.

A Report on the Use of Buccal Tablets of Streptokinase and Streptodornase (Varidase).*†‡

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Enzymes of the hemolytic streptococcus bacteria have been used therapeutically since 1949 for various inflammations, with or without infections. We shall trace this enzyme therapy through the era of topical application, the intravenous and the intramuscular methods of treatment. This present report refers to our experience, using the buccal route of therapy, in which an enzyme is absorbed through the buccal or sublingual mucosa as the tablets dissolve.

The historical background of the development by research of the enzymes that originate from hemolytic streptococcus cultures is a fascinating one and is excellently summarized in a paper by Sherry,[1] which I presume most of you from the St. Louis area heard when it was presented in that city in March, 1958, and which the remainder of you read in the *Annals of Otology, Rhinology and Laryngology*, issue of March, 1959. This article is highly recommended reading, both from the historical and chronological viewpoints, but mainly for the rationale in the use of streptokinase and streptodornase, the enzymes we are discussing.

In 1933, Tillett and Garner,[2] to their surprise, demonstrated that a culture of hemolytic streptococci added to normal human plasma had the capacity of causing immediate clotting, and that then the clotted fibrin immediately liquefied. This was peculiar in that it differed from the usual digestion of solid proteins by a proteolytic enzyme. They found the same

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‡Streptokinase and Streptodornase (Varidase-Buccal Type) supplied by Lederle Laboratories, Division of American Cyanamid Company.

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reaction would take place from cultures obtained from patients, and there were 28 strains of hemolytic streptococci in the cultures used. These infections included scarlet fever, erysipelas, cellulitis, abscesses, and numerous other infections. In 1934 these same authors[3] demonstrated that blood sera from patients having acute bacterial infections are capable of agglutinating certain types of hemolytic streptococci, but this function was present only during the acute infections and disappeared soon after the patients recovered. This function differed from antibody formation in combatting a disease. They reaffirmed that plasma containing a culture of hemolytic streptococci caused immediate agglutination of the organisms in a very few minutes, even in dilute solutions. The inability to produce agglutination in normal serum led to the testing with fibrinogen, which is the protein in plasma, but normally absent from serum. They stated that fibrinogen increased in effectiveness during an acute infection and was the active principle in plasma for the agglutination.

In 1945 Christensen[4] stated that the lysing factor in liquefying a clot represented a proteolytic enzyme normally present in human serum, but inactive. On addition of the hemolytic streptococcus fibrinolysin, the lysing factor (fibrinogen) was activated, similar to the tryptogen, or the enterkinase in other physiological studies relative to the intestinal tract. He also stated that fibrinolysin had no proteolytic activity and that the lysing factor could stimulate the proteolytic activity spontaneously. He postulated that the lysing factor was an inactive proteolytic enzyme, which is a zymogen, in serum activated by a kinase. In this instance the kinase was hemolytic streptococcus fibrinolysin, which he termed streptokinase.

In 1948 Tillett, Sherry and Christensen[5] were still investigating the effects of streptokinase on cultures and specimens of pus obtained from purulent infections, such as pleural exudates; and they discovered that there was a hemolytic streptococcal enzyme in the streptokinase preparation they were using which was capable of causing a complete dissolution of nuclear protein, which is the solid material and sediment in pus. This enzyme was found to be desoxyribonuclease, which they termed streptodornase. This new enzyme, strepto-

dornase, was found to liquefy completely purulent discharge by breaking up all of the nuclei of white blood cells and pus cells. This then led to further studies by Tillett, Sherry and others,[6] reported in 1950, when they announced that streptokinase and streptodornase could be introduced locally and topically into the thoracic cavity to cause rapid enzymatic changes with debridement without any generalized reaction to the patient. Streptodornase was found to have no effect on living cells. The patients who were treated had empyema, pneumothorax, pneumonectomy, osteomyelitis, sinusitis, and degenerative ulcerative lesions.

Of otolaryngological interest, this is the first instance of enzymatic therapy for a sinus infection. The case treated was a 42-year-old male who had a chronic left maxillary sinus infection for three years, with an acute flare-up of ten days' duration. One-half cc. of pus was aspirated from the sinus by a needle, and 500 units of streptokinase and 1000 units of streptodornase were introduced through the needle. A few minutes later 5 cc. of pus had liquefied and, thus, could be aspirated. Follow-up cultures were sterile, and the patient had immediate relief of the acute sinus infection.

The same investigators observed that by topical application of streptokinase and streptodornase there was an acceleration of recovery of patients having any disease in which there was a fibrinous exudate. They noted that extra-vascular clots or loculated hemorrhages quickly cleared up by lysis. Most remarkable was the disappearance of all bacteria from wounds. The removal of debris from these infected areas allowed natural and acquired immunity to function; and, together with antibiotics, which had made their appearance by this time, the recovery was dramatic and rapid. In their studies they found that there was no injury to the mucosa or soft tissues during any local or topical therapy.

In 1952 Johnson and Tillett[7] reported the injection of streptokinase and streptodornase intravenously in animals for dissolving venous thrombi in ears of rabbits, and Sherry and his co-workers[8] in 1954 demonstrated that intravenous injections of streptokinase caused dissolution of thrombi in

the femoral and other large blood vessels in dogs. By 1955 there were several reports[9] of intravenous therapy of streptokinase in humans; however, in the human, this type of therapy was not without complications, such as fever, systolic hypotension of temporary nature, and polymorphonuclear leukocytosis; but, there was no change in the clotting or bleeding time of any of the patients. Extra-vascular bleeding was rare but also was a possible complication during intravenous therapy.

Streptokinase and streptodornase were now recognized as most useful for the treatment of certain and restricted conditions, if used cautiously and a proper blood level maintained, not only in infections but also in injuries where there was extravasation of blood and fluid into the tissues with typical signs of inflammation and edema.

Sherry[1] suspected that many diseases, such as arthritis, gout, bacterial endocarditis, or any of the diseases which are manifested by a precipitation of abnormal material in blood vessels or body tissues may eventually be treated successfully by a specific enzyme.

Soon after the complications were noted following intravenous therapy, attention was directed to the intramuscular method; however, due to localized pain and nodule formation at the site of the intramuscular injection, this method was used sparingly, and mainly in those cases where very few injections were required—and even then only for an acute inflammatory process of short duration. The preparation had been used both intravenously and intramuscularly in animals for infections, inflammations, and prevention of abdominal adhesions; and even to the present date the preparations are still used in general surgery by these methods where there are thromboses and purulent infections.

Since the intravenous and intramuscular methods of therapy had been followed by complications, studies were then begun to ascertain whether another method of administration was feasible or possible. The therapeutic value of streptokinase and streptodornase had been established. The problem was how best to administer it.

After much study for many months it was found by many investigators[10] that there was good absorption of streptokinase and streptodornase by the buccal mucosa, that there was good penetration through the mucosa, and that the desired effect was produced as well by buccal absorption as by the intravenous and intramuscular methods; furthermore, there were no side ill-effects, which had occurred with the other methods of therapy. Various workers experimented with various diseases, including bronchitis, bronchiectasis, retinal vein thromboses, chronic leg ulcers, phlebitis, post-dental extraction sockets, and oro-pharyngeal surgical wounds. In addition, the preparation was used in acute coronary thrombosis, myocardial thrombosis and infarction, thrombosis of various arteries and veins, and other types of disease wherein there was a fibrin clot or exudate, with or without infection. Bruises of muscles and other tissues, sprains of ligaments and tendons were found to respond well. This is of extreme significance because of the economic factor of patients being able to recover quickly and return to work. Athletes, who are continually subject to injuries, with inflammation and hemorrhage, have benefited markedly by this therapy; however, one must not prescribe the preparation for a patient having a collagen disease, a diminished liver function, or a blood-clotting disease. The preparation also should not be administered after or during an acute hemolytic streptococcal disease because of a possible allergic manifestation.

The Otolaryngological Service of Duke Hospital has used streptokinase and streptodornase, buccal type, for various types of cases, but this report is based entirely on my personal experience with my own private patients. In June, 1958, a series of almost 100 cases was started in which we used the buccal method of therapy. The greater portion of these cases reported herein was selected on account of postoperative edema, ecchymosis, hematoma, or infection prior to surgery. We have used the preparation in conjunction with the following operations: external frontal sinus, intranasal ethmoid, sphenoid, submucous resection of the nasal septum, Caldwell-Luc maxillary sinus, shortening of elongated styloid process, removal of pharyngeal and nasopharyngeal tumors, excision of

thyroglossal duct cyst, tonsillectomy and adenoidectomy, and mastoidectomy. We also have used the preparation on patients who have had third molar extractions by their dentists, abscesses of the cheek, hemorrhage and hematoma of the vocal cord, secretory otitis media, hematoma of the tongue (from biting the tongue), herpes labialis, and some of the dermatoses. One case of poison oak cleared immediately, but two cases of trumpet vine (cow itch) did not respond to this therapy. One case of erythema multiforme completely disappeared and did not recur for several months. A case of chronic pharyngitis due to the local use of antibiotic lozenges did not respond, and neither did one case of secretory otitis media; but other cases of secretory otitis media did respond quite well. Two cases of granulation tissue of the mastoid were successfully treated, but in one case granulation of the middle ear did not respond to a minimal amount of the therapy. We really could not evaluate this latter case, however, because of the extreme sensitvity of this patient to every known drug, and treatment had to be discontinued because of stomatitis after using two tablets—our only case of such a complication.

Our most remarkable results relative to rapidity of disappearance of inflammation and infection were observed following external frontal or Caldwell-Luc maxillary sinus surgery. In most instances we were dealing with a purulent frontal sinus infection of osteomyelitic type, or a polypoid and purulent infection in the maxillary sinus. In one case, surgery on the frontal sinus had to be delayed until the patient had buccal and tetracycline therapy because of intense acute infection due to bony perforation with edema, redness, and purulent infection. In another instance there was an unusual frontal sinus disease found unexpectedly at the time of the original operation. This was a case of a giant-cell type of fibrosing osteitis, whereby the entire nasofrontal duct had been sealed off. A new nasofrontal duct was made, a rubber tube inserted, and some weeks later the tube was removed. Then a few months later there was a sudden swelling and abscess formation over the frontal region, with elevation of the periosteum of the frontal bone and eventual perforation

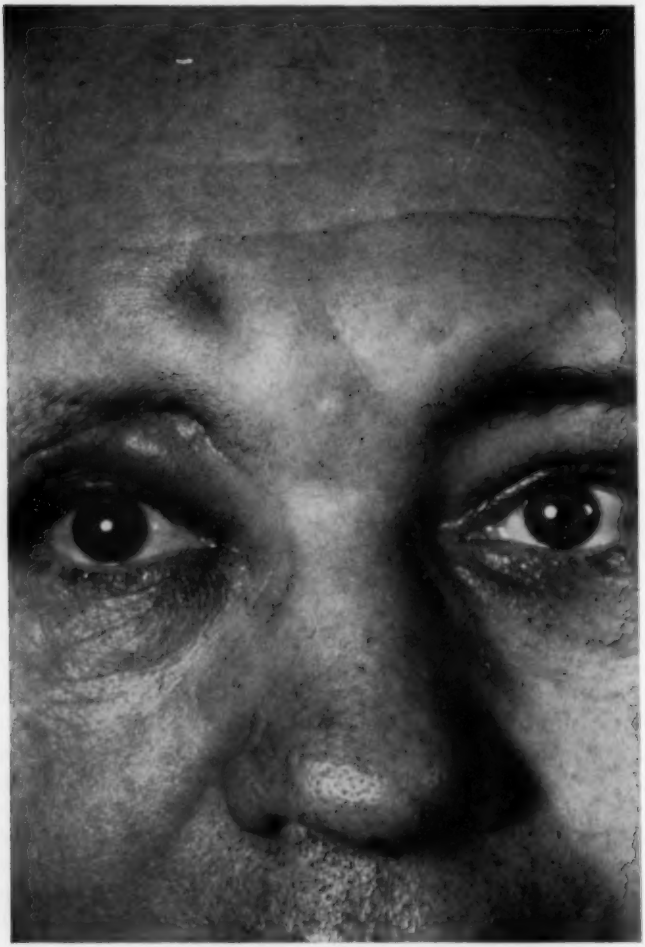

Fig. 1. J.L.A., age 50, D-55658. Had external frontal operation June 9, 1959, at which time ossifying fibroma found—giant-cell type. Nasofrontal duct obliterated. Revised external frontal operation Sept. 16, 1959, following abscess with skin perforation. This photo was made Oct. 10, 1959, and shows complete healing of incision and sinus tract by use of buccal streptokinase and streptodornase with tetracycline.

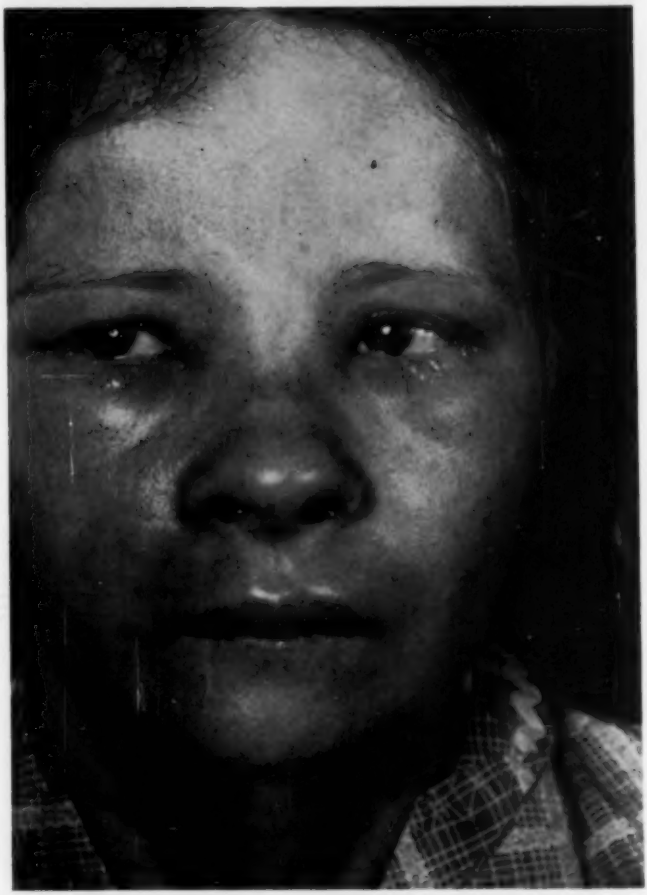

Fig. 2. M.S., age 29, F-1 619. Submucous resection of nasal septum on June 19, 1959. Buccal streptokinase and streptodornase started June 20, 1959. Photo made on June 20, 1959.

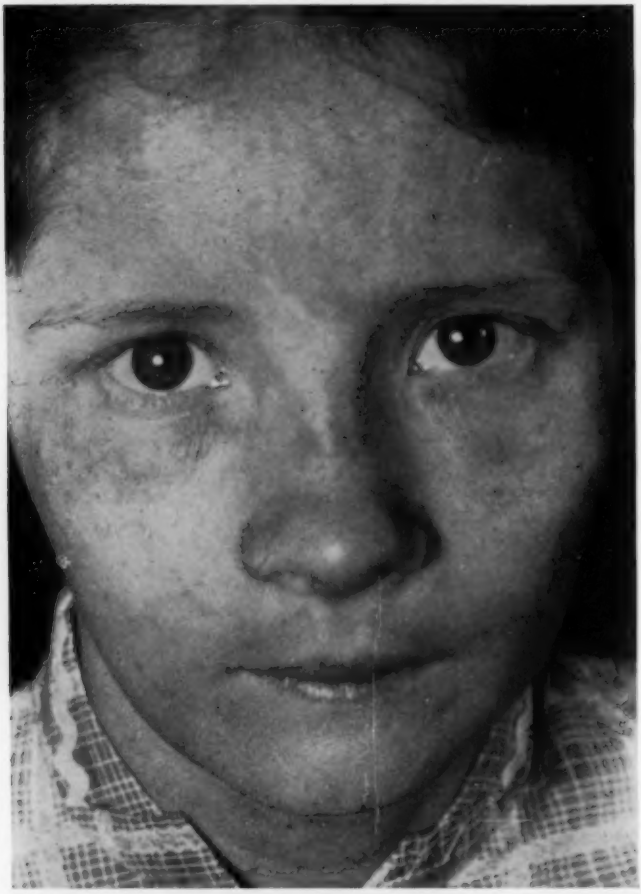

Fig. 3. Same patient, M.S., F-1 619. This photo made on June 22, 1959, after use of ten buccal tablets. Not an allergic individual; unexplained postoperative edema.

of the skin. Again buccal and tetracycline therapy was used, with subsiding of the acute infection (see Fig. 1). Then we were able to proceed with a revision of the external frontal sinus operation, at which time a polyethylene tube was inserted into the re-opened nasofrontal duct. The patient is

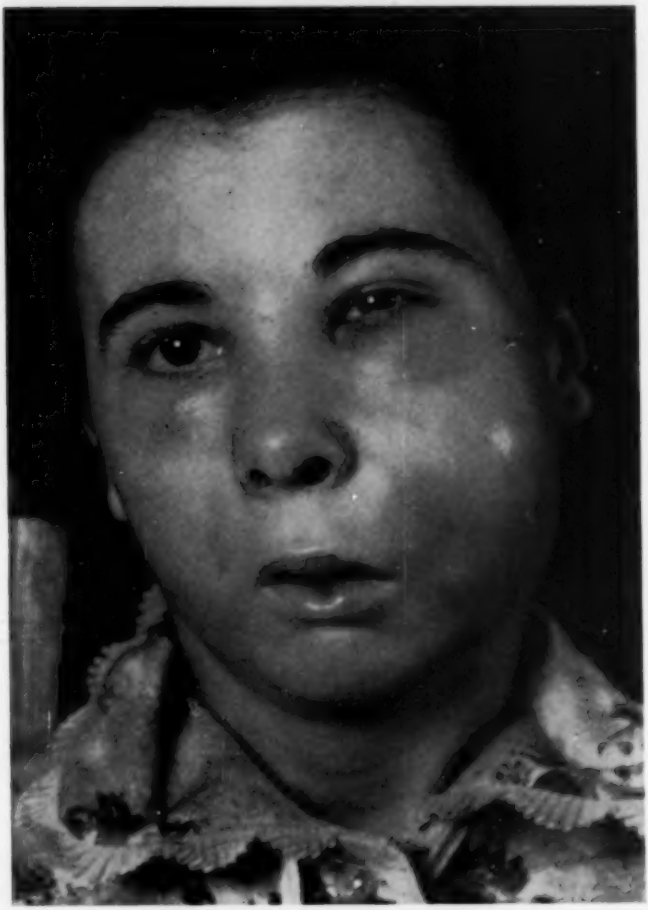

Fig. 4. K.H., age 22, F-04782. Submucous resection of nasal septum Aug. 10, 1959; Caldwell-Luc, left, cysts and polyps found Aug. 11, 1959. Buccal streptokinase and streptodornase started Aug. 12, 1959. This photo made Aug. 12, 1959.

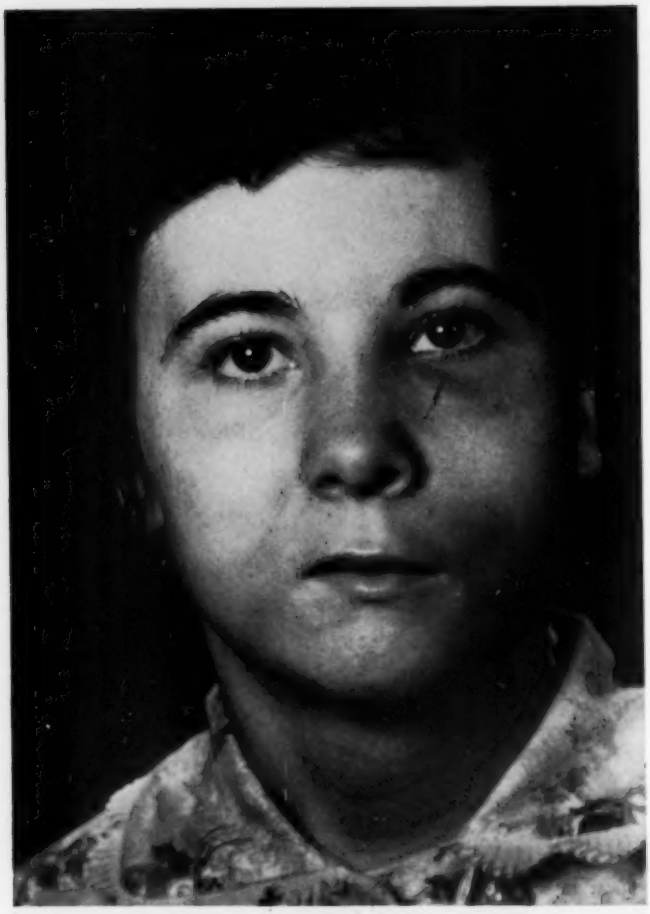

Fig. 5. K.H., F-04782. This photo made on Aug. 15, 1959, after use of 14 buccal tablets.

still wearing the tube *in situ*, and he has had no recurrence of any trouble up to the present time—seven months.

The buccal streptokinase and streptodornase were used in many cases of postoperative hematomata of the nasal septum, after the hematoma had been removed, whether the hematoma

was very small or massive (see Figs. 2, 3). Complete recovery was rapid in all cases except one, a Japanese student who seemed to have no response at all to the preparation. All cases of postoperative maxillary sinus edema and ecchymosis following Caldwell-Luc operation responded with equal rapidity (see Figs. 4, 5). The edema and ecchymosis that occurs, especially in allergic individuals due to stirring up of the infection by the surgery (an example of bacterial allergy), usually subsided in two or three days. This method of therapy produces much more rapid reduction of edema than intravenous calcium therapy or any other means of reducing post-traumatic edema.

I cite a case of herpes labialis, with the lesions extending over the cheek. This patient, a nurse, had recurrent attacks of herpes since childhood, which came on quite often following exposure to sunshine. Smallpox vaccinations and other means of treatment in the past had failed to provide relief. She stated she would like a trial of the buccal therapy and did derive immediate benefit on one day's therapy but continued the medication for several days. There was no recurrence for almost eight months, and then she was able to abort an attack during the recent holiday season by using one tablet.

One patient accidentally bit her tongue early one morning, causing a hematoma about the size of a pencil eraser. By late afternoon, after using only two buccal tablets, the hematoma had completely disappeared. In another patient, a school teacher, a hematoma or submucosal hemorrhage of a vocal cord disappeared overnight following minimal buccal therapy. Ordinarily such a hematoma would require two or three weeks to disappear spontaneously.

We have not used streptokinase and streptodornase in as many post-tonsillectomy cases as we had hoped to; but we can assert that the postoperative slough is less thick and disappears at least one day earlier, and it is also thought there is less pharyngeal angina. We plan to use the buccal therapy on a larger series of adult cases, because we are satisfied that it is beneficial.

It has been established that the optimum dosage of strepto-

kinase and streptodornase for an average-sized adult is four tablets per day. Each tablet contains 10,000 units of streptokinase and at least 2,500 units of streptodornase. The tablets are placed between the cheek and the upper alveolar process. If the patient is edentulous, the tablets are dissolved beneath the tongue. The patient must swallow as little as possible in order to permit complete absorption of the tablets through the buccal mucosa to obtain the full benefits. One, of course, must not drink any fluids as long as the tablet has not dissolved, which generally required 20 to 40 minutes. The tablet must not be chewed or swallowed whole, since there is no benefit to be derived from it after it reaches the stomach. The duration of treatment depends on the type and severity of the condition being treated. In all cases where hematoma or ecchymosis without infection is involved, treatment for two or three days may be long enough. When infection is present, one must continue the treatment for a minimum of five days; and also when infection is present, it is most important that tetracycline drug therapy be used along with the buccal therapy. The tetracycline is administered in full dosage for a minimum of five days also. There are some instances, however, with or without infection, when the therapy must be continued for periods longer than stated above.

SUMMARY.

We have traced the production of a fibrinolytic enzyme, from the hemolytic streptococcus organism, which causes dissolution of blood clots and the melting of exudates and fibrin collections. This enzyme was named streptokinase. It was later discovered that this material also had another enzymatic property which caused liquefaction of pus and other dead cells in any type of discharge, the nuclei of these cells being dissolved by this enzyme, which was named streptodornase.

We have traced the therapy with these enzymes through the topical application method, then the intravenous and the intramuscular methods; and in this paper we have described the buccal method of therapy of streptokinase and streptodornase. We are reporting our experiences with almost 100

cases in which buccal type of therapy was used for various otolaryngological conditions.

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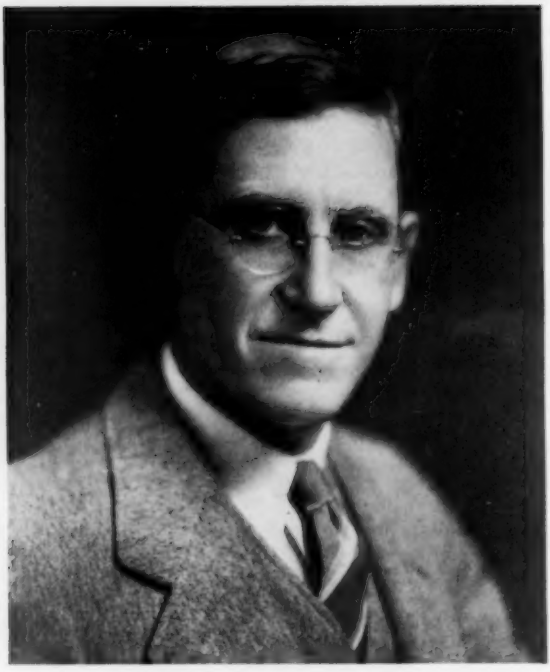
Duke University Medical Center.

HUMBOLDT UNIVERSITY OF BERLIN (CHARITE).

The celebration of the 250th Anniversary of the Charité will be held in Berlin from November 6-19, 1960, in connection with the 150th Anniversary of the Humboldt University.

Applications for participation are to be directed to the Committee for the Preparation of the 250th Anniversary of the Charité, Berlin N 4, Schumannstrabe 20-21, c/o Dozent Dr. med. habil. Dagobert Müller, secretary of the committee.

In Memoriam



GEORGE MORRISON COATES, M.D.,

1874-1960.

Dr. George Morrison Coates died suddenly of coronary occlusion at his home in West Whiteland, Pa., on February 7, 1960, within a month of his 86th birthday. He continued the private practice of his specialty in Philadelphia and the performance of his editorial duties up to the time of his death.

He was born in Philadelphia on March 24, 1874. His Quaker ancestors settled in the area prior to the arrival of William Penn, and members of the family were among the

founders and trustees of the Pennsylvania Hospital, the first hospital to be established in the colonies. His father was variously a cotton broker, textile manufacturer, magazine writer and novelist.

From the University of Pennsylvania, he received a B.A. degree in 1894 and an M.D. degree in 1897. During his college years he was active in sports, serving as captain and manager of the track team and manager of the football team. As a champion bicycle racer, he received numerous trophies and awards.

He engaged in the study of otolaryngology with Dr. Francis R. Packard at the Pennsylvania Hospital and with Dr. Walter Roberts at the Philadelphia Polyclinic and College for Graduates in Medicine from 1901 to 1905. At the latter institution, he became associate professor of otology and, with its merger with the Graduate School of Medicine of the University of Pennsylvania, he was appointed professor and chief of service of otolaryngology in the Graduate School. He was also professor and chairman of the department of otolaryngology in the School of Medicine of the University of Pennsylvania and chief of service at the University Hospital from 1933 until he became emeritus professor in 1939. For many years he was chief of service at the Presbyterian, Abington Memorial, Germantown and Chester County Hospitals.

Dr. Coates became a director of the American Board of Otolaryngology in 1930 and a senior consultant in 1956. He was a past president of the American Laryngological Association, the American Otological Society, the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology and the Philadelphia Laryngological Society. He was the recipient of the Newcomb Award of the American Laryngological Association in 1943, and of the de Roaldes Gold Medal in 1949; the Award of Merit of the American Academy of Ophthalmology and Otolaryngology in 1946, and the Gold Medal Award of the American Otological Society in 1949.

Dr. Coates was chairman of the section of Laryngology, Otology and Rhinology of the American Medical Association in 1927; chairman of the ear, nose and throat section of the

Pennsylvania Medical Society in 1921; chairman of the ear, nose and throat section of the Clinical Congress of Surgeons of North America from 1921 to 1936, and chairman of the ear, nose and throat section of the College of Physicians of Philadelphia from 1928 to 1930.

He was a corresponding fellow of the Societé de Laryngologie des Hôpitaux de Paris and a fellow of the American Bronchoesophagological Association, the American Society of Immunologists, the American College of Surgeons and the Pacific Coast Oto-Ophthalmological Society.

Dr. Coates was editor-in-chief of the *A.M.A. Archives of Otolaryngology* from 1937 to the time of his death; co-editor with Dr. Chevalier Jackson of "The Nose, Throat and Ear and Their Diseases" (1929); co-editor with Dr. I. S. Ravdin of the American Edition of "Kirschner's Operative Surgery" (1937), and editor of the five-volume "Otolaryngology" (1955).

As Assistant Surgeon in the U. S. Navy, he participated in the Battle of Santiago Bay during the Spanish American War, and later served as Lieutenant to Lieutenant Colonel in the Pennsylvania National Guard from 1900 to 1916. During World War I, he served as Major to Colonel, MC, U. S. Army, and was chief of the section of head surgery at the base hospitals in Camp Sevier, S. C., and Camp Hancock, Ga., commanding officer of Base Hospital No. 56, Allerey, France, and president of the Reclassification Board, Bordeaux, France.

Dr. Coates played a major role in the development of graduate teaching in otolaryngology and contributed more than a hundred scientific papers to the literature. His primary interest was teaching and counselling young men in the specialty and his inspiration shaped the careers of many otolaryngologists both in this country and abroad.

Dr. and Mrs. Coates were active in the Episcopal Church and their home in Philadelphia was always a center of hospitality for colleagues and students. Mrs. Coates died in 1954 and his son met a tragic end in 1955, leaving a widow and two sons.

He is survived by his daughter and two grandsons.

H. P. S.

PAN-PACIFIC SURGICAL ASSOCIATION.

Eighth Congress.

Honolulu, Hawaii, September 27-October 5, 1960.

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 27 through October 5 in 1960.

All members of the profession are eligible to register and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities because of limited space.

An outstanding scientific program by leading surgeons promises to be of interest to all doctors. Ten surgical specialty sections are to be held simultaneously.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

COLBY COLLEGE EIGHTH CONSECUTIVE INSTITUTE ON OCCUPATIONAL HEARING LOSS.

Colby College, in Waterville, Maine, announces its Eighth Consecutive Institute on Occupational Hearing Loss, August 8-13. The course is designed to train physicians, plant nurses, plant engineers and others in initiating and conducting hearing conservation programs in noisy industries. The course is comprehensive and includes all phases of the problem. The fee of $200 includes tuition, board and room. Requests for further information should be made to William A. Macomber, Colby College, Waterville, Maine. Dr. Frederick T. Hill and Dr. Joseph Sataloff are directors of the program.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1960.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

President: Dr. J. J. Longacre, 1503 Carew Tower, Cincinnati, O.
President-Elect: Dr. D. C. Samuel Pruzansky, D.D.S., 840 So. Wood St., Chicago, Ill.
Secretary-Treasurer: Dr. Spriestersbach, Ph.D., Department of Otolaryngology, University Hospital, Iowa City, Ia.
Meeting:

AMERICAN BOARD OF OTOLARYNGOLOGY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee Dr., Syracuse 10, N. Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.
Meeting: Palmer House, Chicago, Ill., October, 1960.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President:
Vice-President: Dr. Daniel C. Baker, Jr., 903 Park Ave., New York, N. Y.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3, Pa.
Treasurer: Dr. Charles M. Norris, 3401 Broad St., Philadelphia, Pa.
Meeting: Lake Placid Club, Essex Co., N. Y., May 20, 1961.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, Baltimore, Md.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.
Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Francis W. Davison, Danville, Pa.
Meeting: Lake Placid Club, Essex Co., N. Y., May 21-22, 1961.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Fletcher D. Woodward, 400 Locust Ave., Charlottesville, Va.
President-Elect: Dr. John R. Lindsay, Chicago, Ill.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Treasurer: Dr. K. M. Day, 121 University Pl., Pittsburgh, Pa.
Annual Meeting: Lake Placid Club, Essex Co., N. Y., May 23-24-25, 1961.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Paul H. Holinger, Chicago, Ill.
Vice-Chairman: Dr. Lawrence R. Boies, Minneapolis, Minn.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, Ohio.
Section Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Dean M. Lierle, Iowa City, Ia.
Meeting: Miami Beach, Fla., June 13-17, 1960.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Henry L. Williams, Rochester, Minn.
Vice-President: Dr. Lawrence R. Boies.
Secretary-Treasurer: Dr. James A. Moore, New York City, N. Y.
Annual Meeting: Lake Placid Club, Essex Co., N. Y., May 26-27, 1961.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worcester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Roland M. Loring, 25 E. Washington St., Chicago, Ill.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland 17, Ore.
Annual Clinical Session: Chicago, Ill. (Definite time and place to be announced later).
Annual Meeting: October 1, 1960, Belmont Hotel, Chicago, Ill.

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY.

President: Dr. John J. Conley, New York, N. Y.
Vice-President: Dr. Joseph H. Ogura, St. Louis, Mo.
Treasurer: Dr. F. Johnson Putney, Philadelphia, Pa.
Secretary: Dr. George A. Sisson, Syracuse, N. Y.
Annual Meeting: Palmer House, Chicago, Ill., October 1, 1960.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Oscar J. Becker, Chicago, Ill.
Vice-President: Dr. Sam H. Sanders, Memphis, Tenn.
Treasurer: Dr. Joseph C. Miceli, Brooklyn, N. Y.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28, N. Y.
Meeting: Chicago, Ill., October, 1960.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Walter E. Owen, Peoria, Ill.
President-Elect: Dr. Leland H. Prewitt, Ottumwa, Ia.
Vice-President: Dr. D. A. Skinner, Newark, O.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting: Palmer House, Chicago, Ill., October 8, 1960.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Alberto Gallo.
First Secretary: Dr. Alfredo Martinelli.
Second Secretary: Dr. Guedes de Melo Neto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. Antonio de Almeida, Dr. Gabriel Pôrto, and Dr. Roberto Franco do Amaral.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perellò, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. Gordon H. Francis, 925 W. Georgia St., Vancouver, B. C.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Road, Halifax, Nova Scotia.
Meeting:

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. E. Hartenbower, 203 N. Main St., Bloomington, Ill.
President-Elect: Dr. Edgar T. Blair, Springfield, Ill.
Vice-President: Dr. G. LeRoy Porter, Urbana, Ill.
Delegate at Large: Dr. S. G. Baldwin, Danville, Ill.
Secretary-Treasurer: Dr. C. L. Pannabecker, Peoria, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. George Woodruff, Woodruff Clinic, Joliet, Ill.
Vice-President: Dr. Linden Wallner, 122 So. Michigan, Chicago, Ill.
Secretary-Treasurer: Dr. Robert Lewy, 25 East Washington St., Chicago 2, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. John E. Arthur.
Secretary: Dr. M. L. Battles.
Meetings: First Monday of October through May, University Club, Columbus, O.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. James A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. Dekle Taylor, Jacksonville, Fla.
President-Elect: Dr. Kenneth S. Whitmer, Miami, Fla.
First Vice-President: Dr. William H. Anderson, Jr., Ocala, Fla.
Second Vice-President: Dr. Marion W. Hester, Lakeland, Fla.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr., 1 Davis Blvd., Tampa 6, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Mariano C. Caballero.
Vice-President: Dr. Joseph Freeman.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting: Quarterly in March, May, October and December on the second Thursday of the month, 6:30 P.M., at the McAllister Hotel, Miami, Fla.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Arthur V. Hays.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: May 12-13, 1961, Edgewater Gulf Hotel, Edgewater Park, Miss.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairman in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Petrarca 332-1, Mexico 5, D. F.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY. (Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Ohrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. David D. DeWeese, 1216 S. W. Yamhill St., Portland 5, Ore.
Secretary-Treasurer: Dr. Paul B. Myers, 223 Medical Dental Bldg., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Henry Thiele Restaurant, 23rd and W. Burnside, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. E. H. Campbell, 133 So. 36th St., Philadelphia 4, Pa.
Secretary-Treasurer: Dr. Raymond Jordan, 121 University Place, Pittsburgh, Pa.
Meeting: Palmer House, Chicago, Ill., October, 1960.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. John F. Tolan, 1118-9th Ave., Seattle 5, Wash.
Secretary-Treasurer: Dr. Homer E. Smith, 686 Twelfth Ave., Salt Lake City, Utah.
Meeting: St. Francis Hotel, San Francisco, Calif., May 22-26, 1960.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 No. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place:

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. John J. O'Keefe.
Vice-President: Dr. Joseph P. Atkins.
Secretary: Dr. William A. Lell.
Executive Committee: Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, Dr. William A. Lell, Dr. William J. Hitschler, and Dr. Chevalier L. Jackson.

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Cesar F. Villafuerte.
Vice-President: Dr. Napoleon C. Ejercito.
Secretary-Treasurer: Dr. Eusebio E. Llamas.
Directors: Dr. Antonio L. Roxas and Dr. Armando T. Chiong.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Vice-President: Dr. Carson S. Demling, 513 Jenkins Bldg., Pittsburgh 22, Pa.
Secretary-Treasurer: Dr. Clyde B. Lamp, 8101 Jenkins Arcade, Pittsburgh 22, Pa.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

Meeting: University of Vienna, August 29 to September 1, 1960.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Morris E. Krucoff.
Vice-Chairman: Dr. Max J. Fischer.
Secretary: Dr. Adrian J. Delaney.
Treasurer: Dr. Robert D. Ralph.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: Dr. F. T. Land, 13 Newton Place, Glasgow, C. 3.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow, C. 3.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA
Monterrey 47-201
Mexico 7, D. F.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela.
Treasurer: Dr. Benito Madariaga.
First Vocal: Dr. Rafael González.
Second Vocal: Dr. Juan Oberhauser.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael Garcia Tapia.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DEL NORESTE.

Presidente: Dr. Livio M. Lataza C.
Secretario: Dr. Ramón Prieto.
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Vocales: Dres. Enrique del Buono y O. Benjamin Serrano.

SOCIEDAD VENEZOLANA DE OTORRINOLARINGOLOGIA.

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Secretario General: Dr. Oscar Bustamante Miranda.
Tesorero: Dr. Arturo Marrero Gómez.
Vocales: Dr. Miguel Octavio Russa, Dr. Benjamin Briceño, Dr. Oscar Gonzalez Castillo.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DO RIO GRANDE DO SUL.

President: Dr. Ivo Adolpho Kuhl.
Secretary: Dr. Decio Lisboa Castro.
Treasurer: Dr. Jorge Valentin.

SOCIEDAD PANAMENA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Manuel Preciado.
First Vice-Presidente: Dr. Alonso Roy.
Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. Maria Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Lt. Col. Stanley H. Bear, USAF (MC), USAF Hospital, Maxwell (Air University), Maxwell Air Force Base, Ala.
Secretary-Treasurer: Capt. Maurice Schiff, MC, USN, U. S. Naval Hospital, Oakland, Calif.
Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. L. D. Lide, Florence, S. C.
President-Elect: Dr. Harry Ross, Anderson, S. C.
Vice-President: Dr. David Stack, Spartanburg, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. C.
Meeting: Jointly with the North Carolina Eye, Ear, Nose and Throat Society in Greenboro, N. C. Headquarters will be in the King Cotton Hotel, Sept. 13-15, 1960.

SOUTH WESTERN LARYNGOLOGICAL ASSOCIATION. (United Kingdom)

President: Mr. Michael Robert Sheridan, F.R.C.S., 7 Strangways Terrace, Truro, Cornwall.
Secretary and Treasurer: Mr. James Freeman, F.R.C.S., 22 Downs Park West, Bristol, 6.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. George M. Haik, Professor and Head of the Department of Ophthalmology at Louisiana State University School of Medicine, 812 Maison Blanche Building, New Orleans 16, La.
Chairman-Elect: Dr. Mercer G. Lynch, Assistant Professor of Otolaryngology, Tulane University School of Medicine, 3503 Prytania St., New Orleans 15, La.
Vice-Chairman: Dr. Bernard J. McMahon, Director of the Department and Clinical Professor of Otolaryngology, St. Louis University School of Medicine, 8230 Forsythe Blvd., Clayton 24, Mo.

Secretary: Dr. Albert C. Esposito, First Huntington National Bank Building, Huntington, W. Va. Formerly instructor of Ophthalmology, Ohio State University College of Medicine, Columbus, O.
Meeting: St. Louis, Mo., October 31, 1960, to November 3, 1960.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Va.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Va.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Nime K. Joseph, Wheeling, W. Va.
President-Elect: Dr. John A. B. Holt, Charleston, W. Va.
Vice-President: Dr. William K. Marple, Huntington, W. Va.
Secretary-Treasurer: Dr. Albert C. Esposito, Huntington, W. Va.
Director for Two Years: Dr. James T. Spencer, Charleston, W. Va.